A STUDY ON

ERI GUNMAM

(PEPTIC ULCER)

DISSERTATION

Submitted to

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In Partial fulfillment of the requirements for the award of the degree of

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BRANCH I – MARUTHUVAM

DEPARTMENT OF MARUTHUVAM
GOVERNMENT SIDDHA MEDICAL COLLEGE
CHENNAI - 600 106.

MARCH 2012

CERTIFICATE

This is to certify that this dissertation work on ***"ERI GUNMAM"*** has been carried out by ***Dr S.ARULSORUBI*** during the year *2009-2012* in the ***Post Graduate Department of Maruthuvam, Government Siddha Medical College, Chennai-600106*** under my guidance and supervision in partial fulfillment of regulation laid by **The Tamilnadu Dr. M.G.R Medical University, Chennai** for the *final M.D* **(siddha) Branch I-** ***MARUTHUVAM*** examination to be held in **April 2012**.

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CONTENTS

Introduction

Introduction.

Siddha system of medicine, recognizable among the Dravidian population that prevails in South India and is practiced from time immemorial especially in Tamil populated region. “Siddhars”, Saints and sages of Tamils developed this exceptional system, believed to be in the year of 10000 BC- 7000BC.

“Siddham” means “Arivu” or “complete knowledge”.

“அண்டத்திலுள்ளதே பிண்டம்

பிண்டத்திலுள்ளதே அண்டம்”

ºð¼Ó É¦ »¡Éõ º¦ò¾ÁÕòÐÅ¡í¸î ÍÕì¸õ, Àì¸õ 31.

96 thathuvas were, including Pancha bootha and three Humoural concepts are the fundamental principles of this incomparable system which advocates curative, preventive measures and educates systematized lifestyle through natural way. As herbs, metals, minerals, animals and its derivatives are the core sources for Siddha medicines; this system endorses safest medical care. Siddha is the only system comprising 64 varieties of medicines i.e. 32 classifications of internal medicines and 32 classifications of external medicines.

According to Siddha, “Unavathi seyal marupadu” (changes in Diet and life style), Environmental factors and “Kanma vinai” (congenital/ genetically inherited) are the key factors predisposed for the instigation of each disease via disturbing the equilibrium status of the vital forces termed Vali, Azhal and Ayyam. Noi nadal (to diagnose the affected disease) and Noi mudal nadal (to find out the root cause of the disease) are the crucial approaches employed in Siddha in the process of diagnosis therefore permanent remedy is also achievable since the treatment is focused to cure the disease and root cause of the disease. “Envagai thervu” is the diagnostic tool followed in Siddha for arriving right diagnosis. Vinnavar maruthuvam (Divine method); Manida maruthuvam (Rational method); and Asura maruthuvam (Surgical method) are the main streams of Siddha treatments. Diseases due to Kanma vinai are treated only with DevMaruthuvam.

While treating the patient, special concentration will be paid on Yakkai (Body constitution), Thinai (sort of land where the patient is living) and Paruvakalam (Season) for the effectiveness of the treatment.

Varmam and Thokkanam are exceptional treatment practices existing in Siddha to cure Neuro-Muscular-Skeletal diseases. Yoga-Pranayamam-Dhyanam is advised for chronic diseases and especially psychosomatic in origin. Siddhars developed Kayakarpam methods mainly for longevity with complete freedom from illness and for preventing ageing problems. Siddha not only cures physical, mental diseases but also cares social, moral, and spiritual welfare of an individual".

India is a large country with different cultural and dietary habits, which may produce regional differences in frequency and the natural course of peptic ulcer. **Early observations showed that peptic ulcer was more common among the population of South India than North India.**

A relatively high frequency of peptic ulcer in South India was attributed to the sloppy diet which required little mastication. It was shown that saliva had a buffering capacity and protective effect on the production of peptic ulcer.

Population surveys and the multicentric study conducted by the Indian Council of Medical Research, on the prevalence of peptic ulcer**, the lifetime prevalence of peptic ulcer was 0-61% in Delhi, 0.69% in Chandigarh, and 0.75% in Chennai**

The point prevalence of peptic ulcer in India was 4.72% and the lifetime prevalence was 11-22%.The prevalence of peptic ulcer increased with age, with a peak prevalence of 28.8% in the 5th decade of life.

Peptic ulcer was not only related to socio-economic status. Peptic ulcer (Gunmam) is a disease have seen commonly among white-collar, coolie's, farmer, labour, poor, & rich

During my under graduate study I come across so many patients suffering from dyspepsia, Epigastric pain borborgymes, backpain, vomiting, nausea, &mentaldipperation. With this experience I have selected the disease Eri gunmam for the clinical study of dissertation work on the basis of siddha concepts on course of the disease, diagnosis treatment, and deietic aspects. A wide knowledge of siddha and modern concepts about Aetiology, signs and symptoms. Pathology and Bio-chemical mechanisms will be helpful to conduct the study in a useful manner.

This dissertation is the completion of the data collected during the course of the study for 3 years in my post- graduate, department of maruthuvam, govt. siddha medical college, Chennai-106.

Aim and Objective

AIM AND OBJECT

AIM

The main aim of this study is to do a clinical study in one of the 8 types of gunmam known as Eri gunmam with more interest and observation on the aetiology. Pathology, diagnosis, complications, and the treatment aspects using a time honoured siddha medicines. Such as gunmathichooranam, Musumusukai legium.

OBJECT

To collect and detail the study of various siddha and modern literatures dealing with aetiology, signs and symptoms, diagnosis, prognosis, complications, diet therapy and treatmental aspects of ERI GUNMAM.

- To expose the efficiency of siddha's diagnostic principles.
- To have an idea of the incidence of the disease with reference to sex, age, habit, occupation. Income and social habits of the patient.
- To know the extent of the correlation of definition aetiology, classification, symptom logy, Investigation, diagnostic methods and line of treatment compare with the modern medical system.
- To evaluate the chemical (qualitative & quantitative) properties.
- To evaluate toxicological (acute &sub acute) study.
- To evaluate pharmacological action of the trail drugs.
- To analyse statistical values, and anti microbial studies of all trail drugs.
    - **Gunmathi choornam,**
    - **Musumusukai legium**
- To conduct a clinical trial with the above medicine for **Eri Gunmam.**

Review of Literature

Siddha Aspect

SIDDHA ASPECT

இரைப்பையின் உடற்கூறு

குழ- னுட முனைவந்து தொண்டையப்பா

குறிப்பாக முடியுமென்று தெரிந்து கொண்டு

அழகான அன்னவாகி சொல்ல கேளு

அப்பனே தொண்டையு னடியில் தோன்றி

பழகினதோர் இரைப்பைக்கு செல்லுகின்ற

பண்பாக தடித்ததொரு குழலுக்கே தான்

இனமான அன்னவாகி என்று பேராம்

-அகத்தியர் குணவாடகம் பக்கம் 221

கேளடா இது சுவாச குழலுக்கப்பால்

கெடியாக பின்னியிருக்கும் உண்மைபாரு

நாளடா இதன் நீளம் சொல்ல கேளு

நலமாக ஒரடி தானிருக்கும் சொன்னேன்

வாளடா இதன் வழியாய் அன்னம் முற்றும்

வளமாக சென்றுமே இரைப்பையில் சேர்ந்து

கேளடா சேர்ந்துதான் தேகந் தன்னை

கொற்றவனே வளர்க்குமென்று கொழுமை பாரே

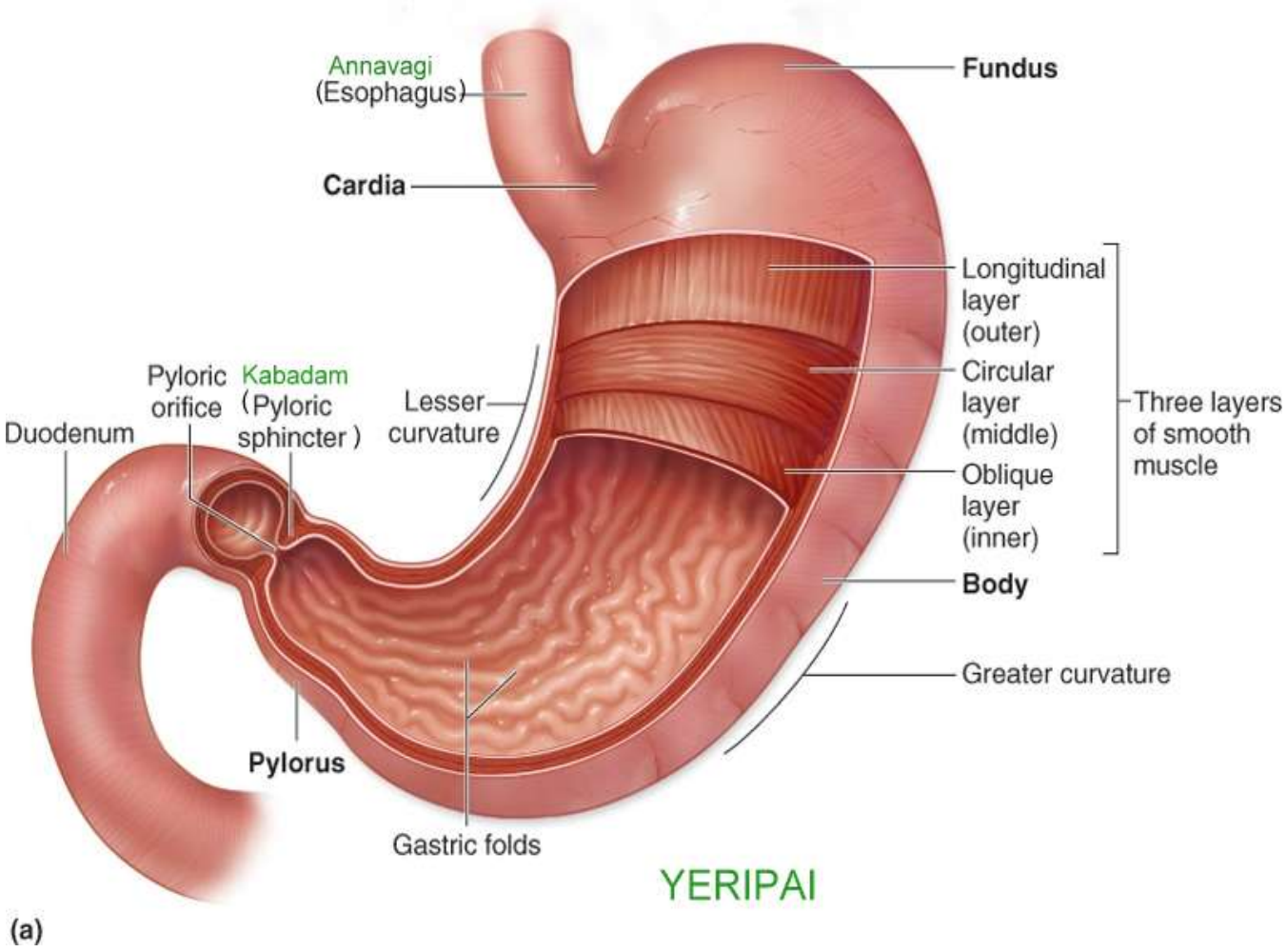
--அகத்தியர் குணவாடகம் பக்கம் 222

VAAI (Mouth)

Mouth is the first part of the GI tract help in ingestion and chewing of food.

ANNAVAGI (Oesophagus)

Oesophagus is a muscular tube that propels the food in to the stomach. it ends in the stomach region.



(a)

கொழுமையுள்ள இரைப்பையின் விவரங்களாம்

குறிப்பான இரைப்பைதான் துருத்தி போலே

வலுவாகி வளைந்துமே இருக்கும் பாரு

வன்மையுள்ள பீ- கா ஸ்தானம் நின்று

பழுவான ஈரல் குழிக்கு மையா

பரிவாக ஒட்டியே இருக்கும் பாரு

தெளிவான சதைகளும் சவ்வும் சேர்ந்து

தேகத்தில் உண்டாகி இருக்கும் பாரு

-அகத்தியர் குணவாகடம் பக்கம் எண், 222

உண்டான இரைப்பைக்கு துவாரமிரண்டு

உத்தமனே இருக்குதடா உறுதிபாரு

நண்டான ஒன்றுதான அன்னவாகியோடும்

செண்டாக சேர்ந்திருக்கும் அதிலே கேளு

செயலான விருத்த சவ்வு மடிப்பொன்றுண்டு

கண்டான கபாடமது ஒன்றுண்டு

கடிதான அதன் குணத்தைக் கூறுவேனே

- அகத்தியர் குணவாகடம் பக்கம் எண், 223

YERIPAI (Stomach)

Stomach is the most dilated part of the digestive tube, and it situated between the end of the oesphagus and the beginning of the duodenum.

The stomach is pear shaped sub division of stomach are fundus body, pyloric antrum, pyloric canal and pyloric sphincter **(Kabadam)**

SIDDHA ASPECT

GUNMAM : Ì ý Áõ

Synonym : Gulmam

Definition:

Gunmam is a clinical entity which depresses both body and mind since it is called as Gunmam.

Gunmam is commonly known as Gastro intestinal disorders usually associated with abdominal pain before or after food with abdominal signs like Indigestion, Nausea , vomiting Gastric eructation, burning sensation in stomch etc.

ETIOLOGY AND PATHOLOGY:

In ancient days Siddhars have recorded the following causes for the manifestation of Eri Gunmam they are Description in

agasthiyar Guru Nadi sasthiram

" Ì ý ÁÁÐ ¾¡ É ØôÒõ Å¢ÀÃ¡ Á ý É¢ø

Ì ¼ø¾É¢§Ä ¸øÖÁ¢¸ ¦¿øÖ ã ì Ì õ

þ ý É Ó¼ý ÅÂ¢ÚôÀ¢ §º¡ ¨ È º¡÷ó¾ì ¸¡ø

ÒÕÅÐ Ì ¼§Ä¡¼ Á¡ Í ÀüÚõ

« ý ÉÁÐ ¦º¡¢ì ¸¡Ð Á¡º¢É¡§Ä

« Ð×ÂÃõ ¯Á¢ã ì Ì ¸¢ÕÁ¢ Òì Ì õ

Å ñ É Ó ¨Ä ì Ì¢Â¢Ä¡§Ä Ì ý Á §Ã¡ ¸õ

Á¡º¡üÈ¡ø Ì ý Áõ ÅÕÅ ¨ ¸¾¡ ý À¡§Ã¡"

agasthiyar Guru Nadi sasthiram Page No: 21

“செய்யான குன்மத்தின் உற்பத்தி தன்னைச்

¦ºôÀ¢¼§Å ÐÅ÷ôÀ¡ É ¦À¡º¢ôÀ¢É¡Öõ

ÁöÂ¡ É Á í ¨ ¸Ô¼ý Á¡÷ì ¸ò¾¡Öõ

Å ¨ ¸Â¡ É ¸¢Æ í Ì Å ¨ ¸ « Õó¾Ä¡Öõ

¯öÂ¡ É Á¢Ç Ì Å ¨ ¸ Ô ¨ ÃôÀ¢É¡Öõ

¯ÚÀº¦¨Â Â¼ì¸¦Î õ Áó¾ò¾¡Öõ

¾öÂ¡É º ñ ¼¡É §¸¡Àò¾¡Öõ

ºÄ¦ôÀ¡Öõ Ì ý Áõ ÅóÐ ¾¡ì Ì õ À¡§Ã"

Yugi chinthamani piranool 800-Page No : 65

Dietetic factors

a) Improper exercise of yogam and pranayamam .

b) Tubers which will produce flatulence .

c) Prolonged starvation,

d) hardly digestible foods.

e) The frequent intake of hot foods..

f) Untimely food.

g) Unbridled sexual indulgence is considered to be the predisposing factors.

PSYCHOSOMATIC CAUSES:

According to **siddha maruthuvanga surukam**

விழியினில் நீரடக்கில்

விதமான இருத்துரோகம்

வழியடு பீநசங்கள்

வந்திடும் நேத்ரரோகம்

அழுகிடும் சிரசில் ரோகம்

அதனுடன் வாதங்கூடில்

பழுதுடல் பண்ணி குன்மம்

பற்றிடுங் குணமுண்டே

சித்த மருத்துவாங்க சுருக்கம் ப,எண், 211

One should not hide his deep sorrow by preventing the tears, such a restricted emotion will results in gunmam.

In Agasthiyar kanma kandam, agasthiyar has stated the psycho spritual reasons for Gunmam as follows

" நன்மையில்லா மனக்கசடு பெருத்த பாவம்

¿ø§Ä¡ ¨Ã ÁÉõ §¿¡¸ô ÀÆ¢ò¾ À¡Åõ

¾ý ¨ÁÂ¢øÄ¡ À¢ü÷ Òº¢ì¸ ¯ ñ ¼ À¡Åõ

º ñ ¼¡Ç ¾òÐÅ§Á ¦ºö¾ À¡Åõ

þ ý ¨ÁÂ¢§Ä þôÀ¡Åõ ÅóÐ Íü È¢

«¾É¡§Ä Ì ýÁ¦ÁÉ ¦Å Îò¾ Å¡§È "

- «¸ò¾¢Â÷¸ýÁ¸¡ ñ ¼õ - Page No: 16.

Teasing elders and polite neighbors and taking food in the presence of starved people is probably the predominant reasons for the sprouting of the awful diseases by their action and covetous mind in the past

TYPES OF CLASSIFICATION & CLINICAL FEATURES

Thirumoolar's View

Thirumoolar classified gunmam into four types as follows,

1. Mega Gunmam
2. Vadha Gunmam
3. Pitha Gunmam
4. Iya Gunmam

1. MEGA GUNMAM

ÅÄ¢ì¸¢ýÈ §Á¸Øõ Å¡Ô×í ÜÊÊø

ÁÄ¢ì¸¢ÁÄºÄõ À¨¾Â¡Áü¸ðÊ Îõ

þÄ¢ì¸¢þ¼ôÒÈõ þÂøÀ¡ö ÅÄôÒõ

ÅÄ¢ì Ìõ ÓÚì Ìõ ÅÕ ï Ý¨Å Ì ýÁ§Á

¾Õ ãÄ÷¸Õì¸¨¼ ¨Åò¾Âõ 600 - Page No : 44

Thirumoolar's view on gunmam is combination of mega with disturbed vayu results in Mega Gunmam . Spasmodic pain in the of left and right side of the abdomen , constipation , etc .

2.VATHA GUNMAM

“ பாருமே வாதமும் வாயுவும் கூடிடில்

° Õ§Á Ì õÀ¢Â¢ø ¯Æý Ú Á¸§È¡ Ì õ

§¸¡ Ì §Á Ì òÐõ Ì¼¨Ä ÓüÚ¸¢Î õ

Å¡Õ§Á Å¡ò¾¢ø ÅÆí¸¢Â Ì ýÁõ

-¾¢Õ ãÄ÷ ¸Õì¸¨¼ ¨ Åò¾¢Âõ - Page No:43

Thirumoolar’s view on gunmam is combination of disturbed vatha and vayu results in vatha Gunmam. The signs and Symptoms are spasmodic pain in the stomach, piercing pain in the intestine, etc.

3. PITHA GUNMAM

" ²üÈ¢É Ì ýÁõ ±Øó¾Å¢¾í §¸Ù

§À¡üÈ¢Â À¢ò¾Óõ Å¡Ô×õ ¦¾¡ó¾ì Ì û

§ºüÈ¢Â « ý Éõ ¦º¡¢ì¸¢ø ÅÄ¢ô§ÀÚõ

Á¡üÈ¢Â ¿¢ÕÈ¢ Å¡ó¾¢Â¡õ À¡Õ§Á.

-¾¢Õ ãÄ÷ ¸Õì¸¨¼ ¨ Åò¾¢Âõ 600- Page No : 43

Thirumoolar’s view on gunmam is combination of disturbed pitha and vayu results in pitha Gunmam . Signs and symptoms are pain in the abdomen after the completion of digestion excessive salivation , vomiting etc .

4. IYA GUNMAM

வழங்கிய ஜயமும் வாயுவும் கூடிடில்

¾Æí¸¢Â « ý Éò¨¾ ºó¾ì¸ ´ð¼¡Ð

ÒÆí¸¢Â « ý Éõ ÒÇ¢ôÒ ¦¸¡Î ò§¾È¢

¯Æí¸¢ ÓÚì¸¢ ¯¼Öõ ÅÄ¢ì Ì §Á

-¾¢Õ ãÄ÷ ¸Õì¸¨¼ ¨ Åò¾¢Âõ 600 -Page No: 44

Thirumoolar’s view on gunmam is combination of disturbed Kapha and vayu results in Iya Gunmam. Fermentation of the swallowed food, accompanied by spasmedic pain in the abdomen and pain in all over the body.

THIRUKANDA MUNIVAR'S CLASSIFICATION

Thirukanda Munivar classified gunmam into eight types but he differs from Yugi Munivar

They are

1 . Vadha Gunmam

2. Pitha Gunmam

3 . Kapha Gunmam

4 . Vatha Pitha Gunmam

5 . Vatha Kapha Gunmam

6 . Pitha Sethma Gunmam

7 . Thrithoda Gunmam

8 . Rattha Gunmam

The Rattha Gunmam is further classified into Rattha Gunmam and Ratha pitta Gunmam

YUGI MUNI'S CLASSIFICATION

Yugi Munivar in his siddha clinical medicine has classified Gunma noi into eight types .

They are

1 . Vayu Gunmam (or) Payuru Gunmam

2 . Vatha Gunmam

3 . Pitha Gunmam

4 . Sethma Gunmam

5 . Eri Gunmam

6 . Vali Gunmam

7 . Satthi Gunmam

8 . Sanni Gunmam

1. VAYU GUNMAM –signs and symptoms :-

"À¡÷ì¸§Å Å¡Ô Ì ýÁõ À¸Ãì §¸Ç¡ö
ÀÕ¸Â§¾¡÷ À¾¡÷ò¾í¸û ¦ºî¡ò¾¼¡Ð
§¾¡÷ì¸§Å Âº É ü¾¡ý ¦ºøÄ¾¡ Ì õ
ÐÕò¾ì Ì û ¸¡üÈÐ§À¡ø ÅÂîÚÓôÒõ
°÷ì¸§Å ÂÊÅÂîÚ ¾É î§Ä ÅóÐ
Á¸ôÒÃ ñ Î Åî øÖô§À¡ø Åî Ì ò¾Ä¡§Á"

-ä¸¨ Åò¾Â ºîó¾¡Á½î

Anorexia, indigestion, bloating of the abdomen, like bellows with increased peristalsis and rigidity in the lower abdomen with sweating, general debility, drowsiness, etc.

2. Vatha Gunmam – Signs and symptoms

" ÅîÕò¾Á¡õ Å¡¾Ì ýÁõ ÅîÇõÀì§¸Ç¡ö
Á¸ò¾¡Û ¿¨¼Ì ¨ÈÔõ ÁÄõ Å¼¡Ð
Å Ì ò¾Á¡ö ¯¼ø¾¡Ûõ Á¸ì ¸Î ì Ì õ
¯È ì ¸Á¡ Î ¾Âì ¸Á¡õ ÔÆ¨ ÄÂ¡ Ì õ
¾Î ò¾Á¡ ï º¡ÃÁÐ ¸É òÐò§¾¡ý Úõ
º í ¨ ¸Â¡õ « ºÉÁ¸ò ¾¡Û ï ¦ºøÄ¡
À Ì ò¾Á¡õ ¦ÀÄì§¸¼¡ö ¨ ¸¸¡ø ´Ôõ
§À¦º¡É¡ ¿¡ÅÈÙõ ¾¨ ÄÒõ §¿¡§Â

- Ô¸¨ Åò¾Â ºîó¾¡Á½î -Page No : 66

Anorexia , constipation , headache, dryness of the tongue , pain all over the body , power diminished in the upper and lower extremities , inability to walk , heaviness of the body , general debility , restlessness etc .

3 . PITHA GUNMAM :- Signs and symptoms

" §¿¡õÀîò¾ Ì ýÁò¾ý ÑðÀí §¸Ç¡ö
ÑÉîÁ ï ºû ¿îÈõ§À¡Ä Ó¸Á¡ Ì õ
Å¡ ï ºò¾ Å¡ó¾Ô ñ ¼¡ö À½ÁÚì Ì õ
ÁÂì¸Á¡ö ¦¿ ï Í¾Éîü §¸¡ ¨Æ¸ð Î õ
ÍÃõ ¦¿ÕôÀ¡ö ¾¡ É îÕì Ì õ ¨ ¸¸¡§Ä¡Ôõ
Í Î ¦ÅÂü ¸ ñ ¼×¼ý ¾¨ ÄÔ ï Íü Úõ

§À¡ ã ò¾Ãõ º¢Åó¾Õì Ì õ ¾¡ ¸ í ¸¡ Ûõ

Ó ì ¸¢§Â ÁÄõ Å¢Æõ ã î Í ñ ¼¡§Á "

-ä ¸¢ ¨ Åò¾Â º¢ó¾¡Á½¢- Page No : 66

Discoloration of the face as yellow , nausea , vomiting , fainting , accumulation of mucous secretion in the lungs , dyspnoea , fatigue of the upper and lower extremities , Giddiness increases when exposure to sunlight , reddish discoloration of urine , increased thirst , constipation etc .

4. Sethma Gunmam:- signs and symptoms

¯ ñ ¼¡ Ì õ Å¡ö¿÷ ¾¡ É¢ ¨ ÇôÒ ñ ¼¡ Ì õ

¯¼øÅüÈ¢ ¸Õò¾Æ¢Ô ÓÃò¾¢ ¨Ãì Ì õ

¦Å ñ ¼¡ Ì õ ¦ÀÄ ý ¦¸ Î ì ¸ Áº É ó ¾û Ù õ

Á¢ì ¸¡É ¾¨ ÄÂ¡¢ì Ì õ ¦ÅÇ¢Õ§Á É¢

¦¾¡ ñ ¼¡ Ì ¦¿ ï Í¾É¢ü Ò¨ ¸îºÖ ñ ¼¡õ

¾¢ Î ì ¸¢ð Î ¿ Î ì ¸ÖÁ¡óò §¾¸ü¾¡Öõ

¾¢ ñ ¼¡ Ì óò ¾¨ Ä¦Â í Ì õ À¡ÃÁ¡ Ì õ

º¢§Äð Î Á Á¡ í Ì ý Á¦Á ý §È ¦ºôÀÄ¡§Á

-ä ¸¢ ¨ Åò¾Â º¢ó¾¡Á½¢ - Page No : 67

Excessive salivation , emaciation , bronochospasm , lowered vitality , loss of appetite fainting , pallor , cough , sudden rigor of the body , heaviness of the head etc.

5 . ERI GUNMAM – Signs and symptoms

¾¢ Î ì Ì Á¡ ±¡¢ Ì ý Áî ¦ºÂ ¨Ä ì §¸Ç¡ö

º¢ÚÅÂ¢üÈ¢ ¦Ä¡¢òÐ§Á Ì ¼ø Ì ÓÚõ

± Î ì Ì õ Å¡ö ¿÷ ÍÃì Ì õ ¾¨Ä ÅÄ¢ì Ì õ

ÅÂ¢Úô Òõ ¸¢Ú¸¢Úò§¾ ²ôÀÁ¡ Ì õ

¦ÅÊì Ì õ ÁÂ¢÷ ¸¡ø§¾¡Úõ Å¢Â÷¨ ÅÂ¡ Ì õ

Á¢¸ô ¦À¡ÕÁ¢ ÅÂ¢Ú ¸Æó ¾¨ ÃîºÄ¡ Ì õ

± Î ì Ì §Á Ì ¼Ä¨ Çì Ì õ þÃí ¸¡ ¾ý Éõ

±¡¢Ô§Á ¯¼¦Ä í Ì Á¢ÕÁ Ä¡§Á.

-ä ¸¢ ¨ Åò¾Â º¢ó¾¡Á½¢-Page No : 67

Burning in the stomach, borborgymes, excessive salivation, headache, bloating, eructation, sweating, Anorexia, diarrhoea, etc.

6. Vali Gunmam

"¾Á¡¸ ÅÂ ê Úóò ¾¨ÃÔ §ÁÉ

¦º¼Ó¨ÄóÐ ¸Õò¾ÆÔ ï º¾Úóò à ì¸õ

ÅÁ¡¸ ÅÂÈ¦ÈóÐ Ó û§À¡§Ä¡ Ì õ

ÅÕò¾Á¡ Âº É Á¸ò ¾¡ Û ï ¦ºøÄ¡

ÓÁ¡¸ Å¡Ä¡Å¾É¡üî ¦º¡Õ¸Ä¡ Ì õ

ÓÐ Ì¾ ñ Î ÅÄ¸¡ Û Á¡Î ôÒ §¿¡Å¡õ

¾Á¡¸ ¸¡ÂÁÐ ¸Î òÐ ì ¸¡ Ï õ

¸É ºÃÁ¡ö ¦À¡öôÀº¡Ôõ ¸¡ Ï ó¾§É"

-ä ¸¦ ¨ Åò¾Â º¡ó¾¡Á½¦-Page No : 68

Bloating of the abdomen, wrinkles in the skin, dryness. Confusion, disturbed sleep Bosborygmus, piercing pain, loss of appetite, pain in the hypochondrium, and pain in the back.

7. Sathi Gunmam – Signs and Symptoms

þÕÁÄ¡ ï ºò¾ Ì ý Áõ Á¡Âø ¨Àì §¸¡Ç¡ö

®ÃÖ ì Ì û ¦ÅÇ¡Â¡ Ì §Á ì¸Á¡ Ì õ

¾¡ÕÁÄ¡óò ¾¡Â ì¸¦Á¡ Î ÁÂ ì¸Ó ñ ¼¡õ

º¡ÚÅÄ¡Ô Ó ñ ¼¡¸¡ Å¡ó¾¡Â¡ Ì õ

¦À¡ÕÁÄ¡õ. ¦ÀÄ ý ¦¸Î ì Ì õ ÁÄõ Å¡¼¡Ð

§ÀÃ¡É « ì¸¡É¡¾¡ ý Á¡¸× ñ ¼¡õ

¦ºÕÁÄ¡õ ¿¨¼Ì ¨ÈÔõ « Õº¡Â¡ Ì õ

º¡Ú ¿Ãõ¦Àø Ä¡õÒ¨¼òÐò ¾¡ÁÕÁ¡§Á'

-ä ¸¦ ¨ Åò¾¡Â º¡ó¾¡Á½¦ - Page No : 68

Burning in the right hypochondric region. Fainting, coma, dull pain accompanied By vomiting, lowered uitality, Constipation. Increased appetite, distaste, prominence of the veins, etc

8. SANNI GUNMAM :- Signs and symptoms

"¦ºôÀÄ¡ ï º ý É¢Ì ý ÁÎ ¦ºÂ¨ Äì §¸Ç¡ö
¾¢Âì¸¦Á¡ Î ÁÂì¸Á¡ö Ì Ç¢Õ ñ¼¡ Ì õ
«ôÀÁ¡õ ÁºÉ Á¢¸ò¾¡ Û ï ¦ºøÄ¡
«ÊÅÂ¢üÈ¢ Ä¢ÃîºøÖÁ¡ö Å¡ö ¿¢ÕÚõ
¯ôÀÄ¡ö- வயிறிழியும் உஷ்ணமாகும்
¯Å÷ì Ì õÅ¡ö ¦¿ ï º¾É¢ø Ò¨¸îºÖ ñ¼¡õ
¦¾ôÀÁ¡ö ãîºÐ×í º¢¨¾ó¦¾ØõÒóò
§¾¸¦Áí Ì í Ì Ç¢÷î º¢ÔÁ¡ Ì õ À¡§Ã"

-ä¸¢¨ Åò¾Â º¢ó¾¡Á½¢. Page No : 67

Fainting , coma , chillness of the body , loss of appetite , borborygmus, flatulenec in the lower abdomen , excessive salivation , diarrhoea , saltish taste , cough , dyspnoea ,etc

CLINICAL FEATURES:-

§ÁÅ¢Â Ì ý Áó¾¡ ý ¦ÉØó¾§¾¡÷ Å¢¾í¸û ¦º¡ø§Å¡õ
À¡¡¢Â À¢ò¾ò §¾¡ Î õ Å¡¾Óõ À¡¢óÐ §º¡¢ø
Å¡¸¢Â Å ñ ñ ¿¢Õõ Å¡ó¾ÂÁ¡ Ì õ À¡§Ã.
À¡ÃôÀ¡ Å¡Ô Å¡¾õ À¡¢×¼§É À¡Å ý È í¸ø
ÃÈôÀ¡ ¿¡Ê ¾ý ¨ É ÅÃ ñ Î¾¡ ý Á¢¸§Å §¿¡ Ì õ
§¸¡ÃôÀ¡ ¦¿ ï º¢ü Ì òÐõ Ì¼¨ Ä ÓÚì¸¢ì ¦¸¡ ñ Î
Å¡ÃôÀ¡ ÅÄ¢ì Ì ¦Áò¾ Å¡¾Á¡ö ÅÆí Ì õ Å¡ö§Å.
ÅÆí¸¢Â «ôÒ§Å¡ Î Å¡¾ ¦Á ý §ÈÂ¡ø
முழங்கிய முறுக்குமேனி திரையும் வதைக்குங்காலே"

-«¸ò¾Â÷ ¨ Åò¾Â¸¡Å¢Âõ-1500 Page No: 29

As per the above poem Agasthiyar says in his Agasthiyar vaidya kaviyam -1500 ‘stated the causes and clinical features of ‘Gunma Noi’.

Flatulence, abdominal pain , indigestion and distention, vomiting,

MUKKUTRA THEORY:

The siddha concept is that, whatever be the course, attribute to the occurrence of "GUNMAM" or any other diseases. The manifestation of the disease is the result of disturbed " Dhoshas" i.e vadha, pitha and kapha.

" ¯üÈ§¾¡ ¯¼Ä¢ý ÜÚ

¯ÚôÒ¼ý Å¢ÃÅ¢ ¿¢ýÚ

ÓüÚ§Á §¿¡ö¸û ±øÄ¡õ

¯¼ø¾É¢ø §¾¡ýÚõ §À¡Ð

ÀüÚ§Á Å¡¾ À¢ò¾

º¢§ÄüÀÉó ¾ý É¢ø µý ¨Èô

ÀüÈ¢§Â §¾¡ýÚõ ±ýÚ

À¸÷ó¾É÷ Ó É¢Å÷ ¾¡§Á"

-« ¸ò¾Â÷ Ì Õ¿¡Ê 235 - Page No: 13

Tastes of the foods have great influence over the physiological activity of three dhoshas, because the tastes and thiri dhoshas are firmed by the different combination of five elements i.e. pancha Boothas The combination of five elements in Thiri Dhoshas are as follows,

1. Vadha (Å¡¾õ) → vali ÅÇ¢ + vinn Å¢ ñ

2. pitha (À¢ò¾õ) → Thee ¾¢

3. kapham (¸Àõ) → Neer ¿÷ + Mann Á ñ .

The elemental combination of tastes are as follows.

1. Sweet (þ É¢ôÒ) = Á ñ + ¿÷
2. Sour (ÒÇ¢ôÒ) = Á ñ + ¿¢
3. Salt (¯ôÒ) = ¿÷ + ¾¢
4. Bitter (¨ ¸ôÒ) = ÅÇ¢ + ¾¢
5. Pungency (¸¡÷ôÒ) = ÅÇ¢ + Å¢ ñ
6. Astrigent (ÐÅ÷ôÒ) = Á ñ + ÅÇ¢

For example the taste sweet is the combination of mann and Neer The Dhosha kapha possesses the same combination, so it is clear that the excess of sweet will initiate kapha and it can be balanced by administering the tastes which consists of the other three boothas.

ERI-GUNMAM : ±¡Ì ýÁõ

¾ Î ì Ì Á¡ ±¡Ì ýÁî ¦ºÂ ¨Äì §¸Ç¡ö
ºÚÅÂüÈ ¦Ä¡òÐ§Á Ì¼ø ÌÓÚõ
± Î ì Ì õ Å¡ö ¿÷ ÍÃì Ì õ ¾ ¨Ä ÅÄì Ì õ
ÅÂÚôÒõ ¸Ú¸Úò§¾ ²ôÀÁ¡ Ì õ
¦ÅØì Ì õ ÁÂ÷ ¸¡ø§¾¡Úõ Å Â÷ ¨ ÅÂ¡ Ì õ
Á¸ô ¦À¡ÕÁ ÅÂÚ ¸Æó ¾ ¨ ÃîºÄ¡ Ì õ
± Î ì Ì §Á Ì¼Ä ¨ Çì Ì õ þÃí ¸¡ ¾ý Éõ
±¡Ô§Á ¯¼¦Äí Ì ÁÕÁ Ä¡§Á.

-ä ¸ ¨ Åò¾Â ºó¾¡Á½. Page No : 67

Burning pain in the stomach , borborygmus ,excessive salivation , headache , bloating , giddiness,eructation , sweating and diarrhoea are the common symptoms in Eri Gunmam

THINAI

Geographically , the living country has been divided into five distinct physical regions , namely :-

1. Kurinchi –Hilly regions
2. Mullai- Forest regions
3. Marutham- Fertile regions
4. Neithal –Sea regions
5. Palai- Sandy regions

Each region has got its own characteristics features which influence the inhabitants, mental , physical , economic , occupational and cultural activities . In each regions on the basis of its peculiar physical and climatic features some ailments are endemic . The preventive and curative measures for these ailments are stated in the medical literature.

KALAM (Seasons)

With reference to the position of the sun in the orbit, the year is divided into six seasons. They are,

1. Kaar kalam – Avanai and Purattasi (August & Septemper)
2. Koothir Kalam –Ippasi and Karthigai (October & November)
3. Munpani Kalam – Markazhi and Thai (December & January)
4. Pinpani Kalam – Masi and Panguni (February & March)
5. Elavein Kalam – Chithirai and Vaigasi (April & May)
6. Muduvenir Kalam – Aani and Aadi (June & July).

In every season there will be changes in the land , water , plants , animals and human beings , which will modify the physiology and making (rendreing) them susceptible to certain specific disease which are common in these seasons . The siddhars have been anticipated those changes and advised certain measures in the form of diet, purgative exercises , etc , to avoid the onset of such ailment .

Uyir Thathu:

Knowledge of three Uyir thathus and seven Udal Kattugal will be helpful to do detailed study on the disease.

Vatham:

It is the life manifestation of Vayu and Ahya boothams. It is mathirai alavu is -1.

Location of Vatham:

Vatham located in the abanan, faces, idakalai, spermatic cord, Pelvic bone, skin, nerves, joints, hairs and muscles.

FUNCTIONS OF VATHAM:

Types of Vatham: It has 10 types;

1. Pranan (Uyir Kaal)

It is responsible for respiration and deigestion. But in **EriGunmam some of patients affected causes Indigestion.**

2. Abanan (Keezhnokku Kaal)

It lies below the umbilicus responsible for the downward expulsion of stools, urine and constriction of anal sptincles. But in **Eri Gunmam some of patients affected causes Diarrhoea and some patients have constipation.**

3. Viyanan (Paruva Kaal)

It is responsible for the action of all organs, sensation and absorption of food. But in **Eri Gunmam some patients affected causes malabsorption**.

4. Uthanan (Melnokku Kaal)

It is responsible for the absorption and distribution of food. In but in **Eri Gunmam some of patients affected causes malabsorption, Nausea, vomiting.**

5. Samanan (Nadu Kaal)

It is responsible for the balancing of the vayus: absorption of nutrition's and water balance of the body. But in **Erigunmam some of patients affected causes indigestion and malabsorption.**

6. Nagan.

It is responsible for the movements for eyelids.

7. Koorman:

It is responsible for the sight, closing of eyelids, yawning and closure of mouth.

8. Kirukaran:

It is responsible for the secretion of mouth and nose, appetite, sneezing, cough. In **Erigunmam some of patients affected lose of apetite**.

9. Devathathan:

It is responsible for aggravating of the emotional disturbances anger,etc. some of patients affected causes stress and strain.

10. Thanajayan:

It escapes from the head on the third day after death.

Pitham:

It is the life manifestation of the bootham. It's mathirai is ½.

Location of Pitham in the body:

Pitham is located in Pirana Vayu, blood, Moolakini, heart, Umbilkal region, abdomen, sweating, saliva, eyes and skin.

Functions of Pitham:

Pitham controls digestion, temperature, vision, appetite, thirst, taste and strength of the body. It is responsible for the formation of red or yellow colour in the body and heat especially during digestion. It is also responsible for giddiness, increase of blood, discolouration of stools, urine, anger, memory and bitter and sour taste.

1. Anala Pitham

Its action is characteristic of thee. This is responsible for digestion of food. In **Eri Gunmam some of patients affected causes like indigestion.**

2. Ranjaga Pitham

It is responsible for the colour and contents of the blood.

In **Eri Gunmam some patients affected causes like anaemia**.

3. Saathagam

It lies in the heart. It is responsible for the action after thinking.

In **Erigunman it is affected causing inability to do work properly**.

4. Prasagam

It is responsible for the complexion of skin.

5. Aalosagam

It is responsible for the vision.

Some patients affected causes defective vision.

Kapham:

It is the life manifestation of mann and Neer. It is mathirai's is ¼.

Location of Kapham:

Kapham is located in Samana Vayu, Sperm, head, tongue, vulva, fat, bone marrow, blood, nose, chest, nerve, bone brain, eyes and joint and it provides the material for the structure of every cell of the body.

Functions of Kapham:

Generally it acts as a distructive factor in the body. When Kapham is in normal condition, it maintains heart function, taste, coolness of eyes, lubricates and aids free movements of the joints.

1. **Avalambagam**

   It causes diseases of the respiratory system when it is affected thereby indirectly affecting the other Iyyams.

2. Kilethagam

Appetite and digestion may not be normal when it is affected.

In **Erigunmam some patients affected causing indigestion and loss of apetite.**

3. Pothagam

It is present in the tongue and gives and taste.

Some patients affected causes like anorexia.

4. Tharpagam

Memory and perception of senses may be affected when this is deranged.

5. Sandhigam

It is present in the joints and helps free movements.

Some patients have mobility of joints is affected due to drying up of the synovial fluid.

SEVEN UDAL KATTUGAL:

There are seven primary body tissues which constitute the entire human body and all the organs of the various systems.

1. Saaram:

It is the end product of digestive process. It gives strength to the body and mind.

It is affected in all patients. They have indigestion.

2. Seneer:

The saram after absorption is converted into seneer. It is responsible for knowledge, strength and health complexion.

In **Eri Gunmam all patients have malabsorption**.

3. Oon:

It gives figure and shape to the body. It is responsible for the movement of the body.

In **Eri Gunmam some patients have loss of weight.**

4. Kozhuyppu

It lubricates the organs and thus facilitates their function.

5. Enbu:

Gives shape to the body helps locomotion and protects vital organs.

6. Moolai / Machai

Present in the bone and it gives strength, maintains the normal condition of the bone.

7. Sukkilam / Suronitham:

Responsible for reproduction.

PINIYARI MURAIMAI:

The method adopted to find out a disease in Siddha is known as PINIYARI MURAIMAI. It is based on the following principles.

- Poriyal Arithal
- Pulanaal Therthal
- Vinavuthal

"Pori" is the five organs of perception namely Nose, Tongue, Eyes, Ears and skin. "Pulan" is the five objects of senses smell. Taste, vision auditory and sensation respectively corresponding to "Pori". Proiyalarithal and Pulanal Therthal go hand in hand with the concept to examining the patients "Pori" and "Pulan" with that of the "Patient's. Pori and Physicians Pulan".

"Vinathal" is a method of inquiring the details of either the patients problem that made him to approach the physician from his own or his/her attendants who accompany them.

Along with, above mentioned principles is also carried out inspection in modern medicine. Besides, Thottuparthal (Palpation) and Thattiparthal (Percussion) are also used to diagnose a patient.

The Primi method adopted to diagnosic the disease is by means of "Envagai Thervugal" (Eight types of investigation), Envagai Thervugal of Physician instruments and can be understood by the following versus.

"நாடிப் பரிசம் நா நிறம் மொழி விழி
முலம் மூத்திரம் மிவை மருந்துவராயுதம்"

– தேரையர் சித்த மருத்துவ நோய்நாடல் நோய்முதல் நாடல் திரட்டு பாகம் –1

ப.எண்.270

Envagai Thervugal Constitute:

1. Naa
2. Niram
3. Mozhi
4. Vizhi
5. Sparism
6. Malam
7. Moothiram
8. Naadi

1. Naa:

The colour character and condition of the tongue change according to the changes of Mukkutram.

In **Eri Gunmam some patients have pallor tongue and some patients have coated tongue.**

2. Niram

Signs of Vatha, Pitha, Kaba, colours, mixed colour cyanosis, Pallor, flusing or yellowish discolouration can be studies by means of Niram.

In **Eri Gunmam some patients have pallor skin due to anaemia**.

3. Mozhi

Constitues high or low pitched voice, slurring and incoherent speech, nasal or crying, hoarseness of voice etc.

4. Vizhi

Along with sight, anatomical lesions are noted. Burning of the eyes, lacrimation, irritation colour change of the eyes also noted.

5. Sparism

By palpation and inspection, the following informtion's were elicited. Temperature of the skin, whether uniformly hot or cold, thickness, fissures stuff / hard swelling, wrinkles, pigmentation of hairs etc.

6. Malam

- Vatha Type : Hard, rough, dry, scanty and black
- Pitha type : loose stools with yellow colour, moderate in quantity
- Kapha type : gray or white coloured stools, huge in quantity with Slimy, mucus and frothy bubbles.

In **Erigunmam some of them have Diarrhoea, and some patients have constipation**.

7. Moothiram

Colour , quantity , froth , thickness , odour , frequency , retention or obstruction signs.

8. Naadi:

Naadi is responsible for the existence of life can be felt one inch below the wrist on the radical side by means of palpation with the tips of index , middle and ring finger , Corresponding to vatham , pitham , Kapham

Three humors vatham, pitham, kabam exists in the ratios 1:1/2:1/4 normally. De arrangement in these ratios leads to various diseases entities.

DIFFERENTIAL DIAGNOSIS

Gunmam should be differentiated from the following chronie diseases of the Gastro intestinal tract which resembles Gunmam.

GUNMA SOOLAI

Yugi Munivar Stated in his work

¾¡ û Ù Ì ý Áî Ý ¨ Ä¾ ¨ É î ¦ º¡ øÄ ì § ¸ Ç¡ ö
¾ÇÕõ ã ò¾ Ã ï º ì ¸ Ä¡ ¸
Å û Ù ÅÂ ü ¦ À¡ ÕÁ º ò¾ Â ¨ Ã î º ø ã ÷ î ¨ º
ÅÄ ò ¦ ¾¡ òÐ î Ý ¨ Ä§À¡ ø ÅÂ üÈ ü § ¿¡ ý È
¦ ¾ û Ù Å¡ ö ¿ Õ È § Äôà ã ñ ¼¡ õ
º Ú òÐ§Á « º É Á ¸ ¦ ÅÐõÀÄ¡ ¸
« û Ù § Á Â í ¸ ¦ ÁÄ¡ ÁÆü º Â¡ Ì õ

ä ¸ º ó¾¡ Á ½ -800- Page No : 71

Constipation, retention of wine, bloating of the abdomen, borborgymes accompanied by vomiting, stabbing pain in the abdomen excessive salivation, gastric evacuation, general emaciation, lower fever, dryness of the body.

VAYITRU SOOLAI (OR) AMA SOOLAI

Yugi munivar described Ama soolai in his work Yugi chinthamani as follows.

ÀÃ×§Á ¬ÁÝ ¨ÄÂ½ý Ì ½óò¾¡ý
À¡Å¡É «ºÃ½ò¾½ý À ñ À½½¡Öü
¾¡×§Á¾ ñ ½÷¾¡ý Ì Êò¾Ä¡Öü
¾Ì ó¾ ÒÇ½ôÒ ¸ºôÒò ¾½ò¾½ôÀ¡Öõ
° ×§Á ¯ÀÅ¡ºõ À ñ ½Ä¡Öõ
¯È ÅÇ÷ó¾ Áó¾¦Á¡ Î º¾ò¾¡Öõ
Å¡×§Á ÅÂ½§È¡ Î Å½Ä¡ô Àì¸í¸û
ÅÄ½òÐ§Á ¸Î ôÒÁ¸ì Ì òÐ ñ¼¡§Á

-ä¸¦ºó¾¡Á½¦-800 - Page No : 71

Indigestion intake of impure water intake of food which are excessive in sour. Bitter and sweet tastes and frequent Starvation the seetham in the Stomach is Vitiated. The vitiated seetham causes dullness in the secretary and modility functions in the stomach. The Vitalized Vatha disturbed the physiological functions of samanakkini and samanavaya as a result of which manifest the pain in the abdomen and the hypochondriam. The pain is pricking in character.

DIAGNOSIS:-

Final diagnosis is made on the basis of the points discussed under the heading clinical features , investigation and the pulse reading of any one of the provocation of Mukkutram . Which are the confirmative signs of the disease “Eri Gunmam”.

Treatment of disease:

After the Thiridoshas are brought down to its equilibrium sate, the signs and symptoms of disease should be treated properly.

For this study.

1. **Gunmathi Choornam** – 1 g 3 times / day with Hot water / milk, After Food.

2. **Musumusukai Lehyam** - 5 gm 2 times / day after food.

Diet:

- As irregular diet is the main etiological factor for Gunman all the patients were chiefly advised to have their food in time.
- They are advised to have well cooked cereals, green leafy vegetables pulses and rice.
- They are advised to get rid of spicy, tubers, food roughage diet, semi cooked and unhygienic diet
- patients were advised to avoid non vegetarian diet.

Habits:

Patients were advised to get rid of smoking, alcohol, etc.

advised to have timely diet.

SPECIAL MEDICINE :-

Yoga Therapy in the treatment of Eri Gunmam

Yoga is a system of siddha philosophy that requires . Intense Mental and physical discipline as a means of attaining union with the unique spirit .

- A system of physical exercise and position used in Yoga .
- A Yogi under guidance of a Guru only goes through eight stages at training as the way to motcha .
- The Yogi taught disciplines and behaviours called Iyama .
- The Yogi taught self purification called Niyama .

The following yoga's are of beneficial effects in the treatment of Gunmam

Virabadrasana

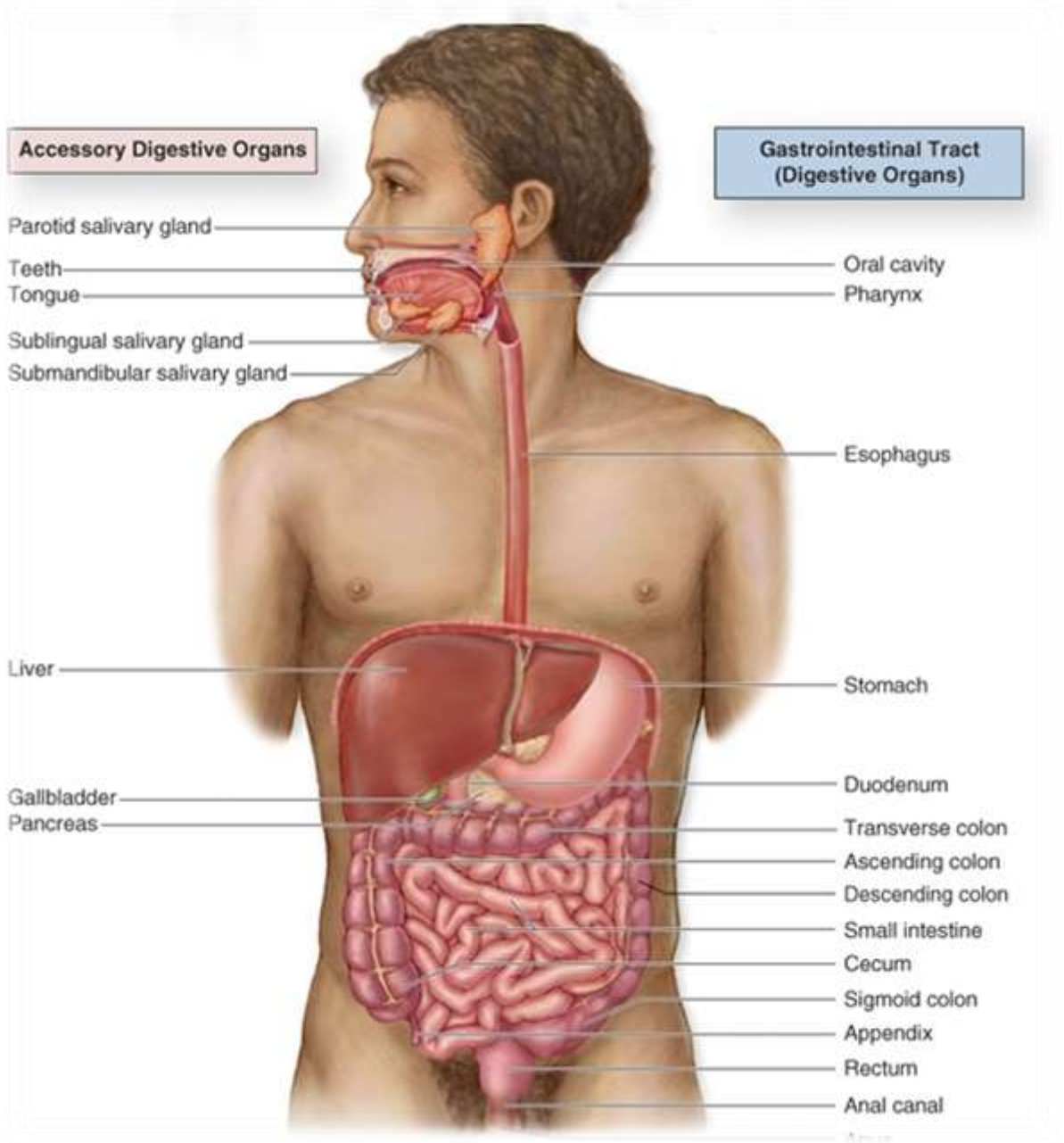
Uradva Prasarita padasana

Jathara Parvarthasana

Savasana

Modern Aspect

G I TRACT


Accessory Digestive Organs
Gastrointestinal Tract (Digestive Organs)
Parotid salivary gland
Teeth
Tongue
Sublingual salivary gland
Submandibular salivary gland
Liver
Gallbladder
Pancreas
Oral cavity
Pharynx
Esophagus
Stomach
Duodenum
Transverse colon
Ascending colon
Descending colon
Small intestine
Cecum
Sigmoid colon
Appendix
Rectum
Anal canal

STOMACH

Anatomy

The word stomach is derived from the Latin stomachus which is derived from the Greek word stomachos, ultimately from stoma "mouth". The words gastro- and gastric (meaning related to the stomach) are both derived from the Greek word gaster).

The stomach lies between the oesophagus and the duodenum (the first part of the small intestine). It is on the left upper part of the abdominal cavity. The top of the stomach lies against the diaphragm. Lying behind the stomach is the pancreas. The greater omentum hangs down from the greater curvature.

sphincters

Two sphincters keep the contents of the stomach contained. They are the esophageal sphincter (found in the cardiac region, not an anatomical sphincter) dividing the tract above, and the Pyloric sphincter dividing the stomach from the small intestine.

The stomach is surrounded by parasympathetic (stimulant) and orthosympathetic (inhibitor) plexuses (networks of blood vessels and nerves in the anterior gastric, posterior, superior and inferior, celiac and mysenteric), which regulate both the secretions activity and the motor (motion) activity of its muscles.

In adult, the stomach has a relaxed, near empty volume of about 45 ml. Because it is a distensible organ, it normally expands to hold about one litre of food,[4] but can hold as much as two to three litres. The stomach of a newborn baby will only be able to retain about 30 ml.

The stomach is divided into four sections, each of which has different cells and functions.

The sections are:

Cardia

Where the contents of the oesophagus empty into the stomach.

Fundus

Formed by the upper curvature of the organ.

Body or Corpus

The main, central region.

Pylors

The lower section of the organ that facilitates emptying the contents into the small intestine.

Peritoneal folds attached to stomach

Gastro phernic ligaments, gastro splenic omentum, greutes omentum and lesser omentum, parasympathetic fibres reach through the gastric nerves. The anterior gastric nerve, the branch of the left vagus descends to the anterior surface of stomach close to the lesser curvature. The posterior gastric nerve a branch of right vagus runs close to the lesser curvature on the posterior surface of stomach.

Like the other parts of the gastrointestinal tract, the stomach walls are made of the following layers, from inside to outside

Mucosa

The first main layer. This consists of the epithelium and the lamina propria (composed of loose connective tissue), with a thin layer of smooth muscle called the muscularis mucosae separating it from the submucosa beneath.

sub mucosa

This layer lies over the mucosa and consists of fibrous connective tissue, separating the mucosa from the next layer. The Meissen's plexus is in this layer.

Over the sub mucosa, the muscular is external in the stomach differs from that of other GI organs in that it has three layers of smooth muscle instead of two.

Inner oblique layer: This layer is responsible for creating the motion that churns and physically breaks down the food. It is the only layer of the three which is not seen in other parts of the digestive system. The antrum has thicker skin cells in its walls and performs more forceful contractions than the fundus.

Middle circular layer: At this layer, the pylorus is surrounded by a thick circular muscular wall which is normally tonically constricted forming a functional (if not anatomically discrete) pyloric sphincter, which controls the movement of chyme into the duodenum. This layer is concentric to the longitudinal axis of the stomach.

Outer longitudinal layer: Auerbach's plexus is found between this layer and the middle circular layer.

Serosa

This layer is over the muscular is externa, consisting of layers of connective tissue continuous with the peritoneum.

Blood supply

left and right gastric artery,
left and right gastroepiploic artery
short gastric artery.

The lesser curvature of the stomach is supplied by the right gastric artery inferiorly, and the left gastric artery superiorly, which also supplies the cardiac region. The greater curvature is supplied by the right gastroepiploic artery inferiorly and the left gastroepiploic artery superiorly. The fundus of the stomach, and also the upper portion of the greater curvature, are supplied by the short gastric artery.

Like the other parts of the gastrointestinal tract, the stomach walls are made of the following layers, from inside to outside:

NERVES SUPPLY

The anterior gastric nerve, the branch of the left vagus descends to the anterior surface of stomach close to the lesser curvature. The posterior gastric nerve a branch of right vagus runs close to the lesser curvature on the posterior surface of stomach.

The sympathetic nerves are a) Vasomotor, b) Motor to pyloric sphincter, but inhibitory to the other parts of the gastric musculature and c) main pathway for main impulses from the stomach.

Parasympathetic supply to stomach are (posterior gastric nerve). Right vagus (2) Coeliac branch (Gastric Branches).

Applied Anatomy:

The gastric ulcers are common in the lesser curvature but more in the pyloric region.

a) Vessels to the pyloric end of the stomach carry less blood compared to their size, since they are branches of the hepatic than to the left gastric and they branch to the stomach from the splenic. The difference in the blood supply as regarded as one of the factors responsible for the occurrence of larger percentage of gastric ulcers towards the pyloric end of the mucosa.

b) There is no sub mucosal plexus. The occurrence of this type i.e. direct from the sub-serous vessels apparently increases from cardiac to the pyloric regions in man. Increased vagal tone can produce marked constriction of the mucosal vessels of this region causing ischemia and necrosis.

c) Further musculature of the pyloric end is thicker and more powerful.

d) Anterior-venous anastomoses occurs in the gastro-duodena mucosa and dysfunctions are these might lead to local ischemia and ulcer formation.

A chronic gastric ulcer usually occurs in alkaline producing mucosa i.e. pyloric antral mucosa closer to the lesser curvature. The secretion of acid and pepsin is by the mucosa of the body and to a lesser extent, the fundus and it is controlled by a) vagus and b) the hormone gastric produced by the antral mucosa. For treatment of chronic gastric ulcer the acid secretions are reduced by a) the section of the vagus nerve and b) by removing the gustrin producing pyloric and run.

A chronic ulcer can perforate through the anterior wall of stomach into the greatest sac producing diffuse peritonitis. If the ulcer perforates the posterior gastric wall, it open into lesser sac and may erode the pancreas to produce severe back pain or bleeding of the splendid artery. Pain from the stomach is referred to the epigastrium. The afferent pain impulses pass through the sympathetic the coeliac ganglia and reach the spinal cord through the greatest splanchnic nerve.

The interior of the stomach can be viewed directly or mucosal biopsy can be done through an Endoscope. Endoscope's purpose is to differentiate between gastric or duodenal ulcer and cancer of the stomach through HPE.

Duodenum:

Duodenum is the most fixed part of the small intestine. It extends from the pyloroduodenal junction, 2.5 cm to the right of the midline on the transpyloric plane to the duodeno jejuna flexure situated on the left side of the second lumbar vertebra.

Situation and Shape:

It is situated on the posterior abdominal wall and is in the form of letters 'C' the concavity of its curvature is directed upwards and to the left.

Measurements:

Its length is 25cm and width is about 3.75 cm. it is the widest part of the small intestine.

RADIOLOGICAL ANATOMY:

The alimentary tract can be demonstrated radiologically by giving barium meal a watery suspension of barium sulphate and taking X-ray pictures at regular intervals. The fundus of the stomach, the lesser and greater curvature and the incisura angularis can be easily made out.

The barium meal passes into the first part of the duodenum and forms a homogenous triangular shadow called the duodena' cap, its base being directed towards the pylorus. The duodenal cap shadow is smooth due to the absence of mucous fold. Persistent deformity of the duodenal cap is characteristic of duodenal ulcer.

Physiology of the Alimentary Tract:

The alimentary tract is a co-ordinated structure with the function of ingesting and absorbing nutrients and excreting unabsorbed waste products. It should not be regarded as a series of separate organs. Since the role of each component is closely related to that of other parts of the tract. Its operation may be considered under the following heading.

1.Motility:

Apart from the striated muscle in the upper oesophages, smooth muscle is responsible for the motility of the gastrointestinal tract. The smooth muscle produces 'slow waves' which are conducted over long distances. These do not result in contraction but they enable contractions in different areas to be co-ordinated.

2. Defence Mechanisms:

These are necessary to protect the mucosa from its own digestive enzymes and from the bacterial population to which it is exposed. These mechanisms include a rapid turnover of the epithelial cells, the production of mucous and a specialized immunological system.

3. The controlling and Co-ordinating Mechanisms:

The cells of the gastrointestinal tract are controlled by the transmission of chemical messenger which are amino acids or their derivatives, amines or peptides. The transmission of the message is called neuro crine when it is across a synapse, when it is through the intercellular space it is called as pericrania and when it is via the blood it is called as endocrine. Many harmones and peptides have now been discovered. Each being produced by a distinctive cell type in the gastro intestinal mucosa of pancreas peptide hormones or pericrania substances are released in response to the food in the upper gastrointestinal truct, their function being to control the progression of nutrients by modifying nervous stimuli, and to stimulate of inhibit, secretions at the appropriate time. It is now recognized that each even is influenced by a multitude of nervous and hormonal stimuli. For example, the secretion of acid by the stomach is produced mainly by vagal stimuli and the release of gastrin, but many other enteric hormones probably have smaller roles to play.

4. Secretion:

The secretion of enzymes and detergents enables protein, carbohydrate and fat to be digested before absorption. The secretion of electrolytes provides the correct PH for each stage of digestion.

5.Absorption:

The absorptive system consists of specialized cells, together with the portal venous system and lymphatics.

Small Intestine:

Here the co-ordination is due to the slow wave in the longitudinal muscle fibres. It is the pacemaker which dictates the times at which any given segment of the gut can contract. The frequency of the slow wave in the duodenum is greater than in the ileum, thus enabling the prominal bowel to override more distal areas.

1. Secretion:

The production of secretions for the digestion of nutrients is under nervous and hormonal control.

2. Gastric Secretion:

In response to the sight or smell of food, the vagus stimulate acid and pepsin secretion by a direct effect on the parietal and peptic cells. It also initiates the release of gastrin from the antrum. More sustained output of this hormone is produced by a rise in PH and by ingested protein. Gastrin then enters the blood stream and acts on the body of the stomach to produce acid and pepsin to digest the protein. It stimulates acid secretion through the release of histamine. Some mechanisms are also involved to turn off gastric secretion, once digestion within the stomach is complex. They are

largely the same as those which slow gastric emptying i.e. The release of the enteric hormones, secretin and cholecystokinin, and also the presence of a low PH in the gastric antrum which inhibits the further release gastrin.

3. Pancreatic Secretin:

Bile and intestinal secretins, acid, fat and hypertonic solutions in the duodenum release the hormones secretin and cholecystokinin from the duodenal mucosa into the blood stream. Secretin stimulates the acinar cells of the pancreas to produce bio carbonate which neutralizes the gastric acid and provides a neutral PH for the activation of the pancreatic enzyme lipase, amylase, and trypsin. They are produced in response to cholecystokinin, which causes contraction of the gall bladder so that an adequate supply of bile acids reaches the intestine when fat has to be digested the enteric hormones are responsible for the secretion of succus entericus by the mucosa of the small intestine, which contains bicarbonate and additional enzymes.

4. The absorptive system:

The area the absorption in the small intestine is increased several hundred fold by the presence of villi and microvilli. The surface of the individual cells are made of microvilli which passes a multitude of enzyme systems for the final stage of digestion of nutrients followed by their absorption. In celiac disease and topica sprue and the surface area of the small intestine is reduced because of the atrophy or loss of villi, and malabsorption results.

5. Defence Mechanisms:

Cell Turn over:

The epithelial cells of the gastrointestinal tract are constantly renewed. for example, the epithelial surface of the small intestine is replaced for every 48 hours. The desquamated cells are digested and their products are reabsorbed. In the intestine, cellular turnover has been shown to be slower, than in normal germ free animals and it can be argued that this turnover to some extent provides protective mechanism.

Production of Mucous:

Mucous producing cells are present throughout the gastro intestinal tract and mucous has a protective function. In the stomach, the mucous layer on the surface of the epithelium contains bicarbonate ions which form part of the barrier to gastric acid.

Immunological system:

The lamina propria of the stomach and the intestine contains many lymphocytes and plasma cells. Some of these cells sysnthesise secretary Ig A which is resistant to digestion by intestinal enzyme and has a role in protecting mucosal surface from bacterial invasion. It is thus of particular importance in the small intestine where bacterial colonization is deleterious.

THE SYMPTOMS OF ALIMENTARY DISEASE:

Pain is often the most important symptom of gastrointestinal disease. It must be analysed in relation to its main site, radiation character, severity, duration, frequency, time of occurrence, aggravating and relieving factors and any associated phenomena. The characteristics of abdominal pain are often diagnostic for example in peptic ulceration and acute appendicitis.

1. Heartburn

Burning retrosternal sensation due to reflex esophagitis.

2. Water brash

The sudden filling of the mouth with Saliva which is produced as a reflex response to a variety of symptoms from the upper gastrointestinal tract, e.g. peptic ulcer pain.

3. Vomiting

May occur in diseases of the stomach or intestine. Vomiting of large quantities of food and secretions late in the day or night indicates gastric outlet obstruction. Vomiting which relieves pain is often due to a peptic ulcer.

4. Loss of appetite

May be a local cause such as carcinoma of the stomach, but may also be a feature of any debilitating disease or due to psychological disturbance.

5. Regurgitation

Appearance of previously swallowed food in the mouth without vomiting. It usually has an acid or bitter taste because of the presence of gastric juice or bile but not in patients with obstruction in the oesphagus.

6. Dysphasia

Difficulty in swelling.

7. Flatulence

Due to excessive swallowing of air (aerophagy) which in turn may be due to anxiety under normal circumstance a small amount air may be expelled as a belch. The remainder passes into the intestine. Some will be absorbed but most, particularly the nitrogen will be expelled per rectum.

8. Constipation and Diarrhoea

Sometimes difficult to define.

9. Loss of weight

May be due to a reduced intake of food because of anorexia nausea or vomiting to malabsorption of nutsients or to the loss of protein from a diseased bowel as in ulcerative colitis carcinoma is the most important alimentary cause of loss of weight.

10. Anemia

Usually occur in massive hemorrhage or in a non-observed passage of tarry stools.

PEPTIC ULCER

Definition:

The English word "peptic" comes from the Latin word pepticus which comes from the Greek word peptikus which comes from the Greek word peptein, meaning "to digest". The English word "ulcer" comes from the Latin word ulcus (genitive: ulceris), meaning "a sore, a wound, an ulcer".

The term 'Peptic Ulcer' refers to an ulcer in the lower oesophagus, stomach or duodenum, in the jejunum after surgical anastomosis to the stomach. Or rarely in the ileum adjacent to a Meckel's diverticulum, ulcers in the stomach or deuodenum may be acute or chronic, both penetrate the muscularis mucosae but the acute ulcer shows no evidence of fibrosis. Erosions do not penetrate the muscularis mucosae.

AETIOLOGY

Mucosal resistance against Acid – Pepsin

The immediate cause of peptic ulceration is digestion of the mucosa by acid and pepsin up the gastric juice, but the sequence events leading to this is unknow. Digestion by acid and pepsin cannot be the only factor involved, because the normal stomach is obviously capable of resisting digestion by its own secretions. The concept of ulcer aetiology may be written as acid plus pepsin versus mucosal resistance. Some factors which affect this balance can be identified.

Gastric Hypersecretion

Ulcers occur only in the presence of acid and pepsin; they are never found in achlorohydric patients such as those with pernicious anemia. On the other hand severe intractable peptic ulceration nearly always occurs in patients with the zollinger – Elisson syndrome which is characterized by very high acid secretion. Acid secretion is more important in the aetiology of duodenal than gastric ulcer, because patients with duodenal ulcer, as a group, secrete more hydrocholoric acid normal individuals.

Mucosal Resistance

Several mechanisms protect the gastric mucosa from hydrogen ions secreted into the lumen of the stomach. The surface epithelial cells secrete bicarbonate ions which creates an alkaline milieu at the surface of the mucosa; this bicarbonate secretion is under the influence of mucosal prostaglandins. The fight function between the epithelial cells and their surface lipoprotein layer provide a mechanical barrier. The normal turnover of epithelial cells and gastric mucous also has a

protective function, collectively, all these mechanisms can be described as the 'Gastric mucosal barrie'. Its integrity is important in preventing gastric ulcer and some of these mechanisms may also operate in the duodenum.

Factors Reducing Mucosal Resistance

Several drugs particularly those used in Rheumatoid arthritis will disrupt the gastric mucosal barrier when aspirin is in solution at a PH below 3.5, it is undissociated a and fat-soluble, so that it is absorbed through the lipoprotein membrane of the surface epithelial cells; during absorption damages the membrane and the tight junctions. It also inhibits prostaglandin synthesis thus reducing bicarbonate secretion by the surface epithelial cells. Aspirin has been shown to be an important actiological factors in gastric ulcer. There is also a relationship between aspirin ingestion and acute bleeding from the upper gastrointestinal tract.

Reflux of bile and intestinal secretions into the stomach occurs more frequently in patients with gastric ulcers than in normal individuals or patients with duodenal ulcer, due presumably to a poorly functioning pyloric sptineter. Bile damages the gastric mucosal barrier, predisposing the mucosa to ulceration chronic gastritis is more common in patients with gastric ulcer and it may be caused by damage from regurgitated bile and intestinal secretions..

Diet

The lack of protein deficient diet and untimely meals in these refined food resulting in a failure to gastric acid ingestion of refined cereals is the prominent factors in the increased incidence of duodenal ulcer.

Smoking, Alcohol

Incidence of peptic ulcer is high among smokers than among non-smokers. Gastric ulcer tend to heal more rapidly in patients who step smoking than in those who do not smoking decreases the therapy. All these facts suggest that it is an actiological factor in the development of peptic ulcer. Gastric ulcer commonly occurs in association with alcoholic cirrhosis.

Heredity

Patients with peptic ulcer often have a family history of the disease. This is particularly the case with duodenal ulcers which develops below the age of 20years. Gastric and duodenal ulcers are inherited as separate disorders; thus the relatives of gastric ulcer patients have three times the expected number of gastric ulcers but duodenal ulcer occurs with the same frequency amongst relatives as in the general population.

Stress induced ulcers

People who are highly nervous and emotional and who worry, fear and feel anxiety are particularly susceptible. These emotional and nervous factors in turn may lead to hyper secretion and hyper mobility of the stomach the nervous control of the vascular system in the gastric on duodenal walls may be so disturbed that there is diminision in the blood supply to the mucosa of the stomach and duodenum making its susceptible to acid secretion.

Helicobacter pylori

A major causative factor (60% of gastric and up to 90% of duodenal ulcers) is chronic inflammation due to Helicobacter pylori that colonizes the antral mucosa. The immune system is unable to clear the infection, despite the appearance of antibodies. Thus, the bacterium can cause a chronic active gastritis, resulting in a defect in the regulation of gastrin production by that part of the stomach, and gastrin secretion can either be decreased (most cases) resulting in hypo- or achlorhydria or increased. Gastrin stimulates the production of gastric acid by parietal cells and, in H. pylori colonization responses that increase gastrin, the increase in acid can contribute to the erosion of the mucosa and therefore ulcer formation.

Drugs

There is much suggestive evidence that treatment with aspirin. Phenylbutozone etc. may aggravate peptic ulcer incidence.

Blood Groups

Peptic ulcer tends be more common in people with blood group ‘O’ Gastric ulcer tends to be more common in people with Blood Group ‘A’

Association with other Diseases

Peptic ulcers in association with almost all diseases, the incidents is noted in patients with Achlohydia namely pernicious Anaemia and Atrophic Gastritis Gastric Carcinoma, Diapharagmatic, Herinia, Duodenal stasis, emphasema, corpulsmonale and Rheumatoid disease, Cirrhosis of liver, Tuberculosis.

Occupational Factors

The occupational survey carried out by hussian from Hyderabad reported that 60% duodenal ulcer cases were in farmers. It may be trace that peptic ulcer is commonamong south Indian agriculturists. It is also common is executives, doctors and industrialists.

Pathology

Chronic gastric ulcer is usuallysingle; 90% are situated on the lesser curve within the antrum or at the junction between body and antral mucosa, chronic duodenal ulcer is usually in the first part of the duodenum just distal to the junction of pyloric and duodenal mucosa; 50% are on the anterior wall, more than one peptic ulcer is found in 10 – 15% of patients acute ulcers on erosions are frequently multiple, and are more widely distributed.

Clinical Features

A duodenal ulcer follows a chronic course for upto 20 years and while the treatment with histermine H2 – receptor antagonist drugs may effect prompt healing, there is no evidence that the natural history of the ulcer is affected. The course of gastric ulcer is probably less chronic. While there are good grounds for believing that gastric and duodenal ulcers are different deceases it is convenient to describe the general features of "Peptic Ulcer" as inclusive of both, there is no difference in their occurrence.

Peptic ulcer may be present in different ways. The commonest is chronic, episodic pain extending over months on years. However, the ulcer may come toa attention as an acute episode with bleeding or perforation, with little or no previous history, occasionally the patient presents with the symptoms of gastric outlet obstruction, having negligible trouble previously.

Pain is the characteristic symptoms of peptic ulcer, and it has three notable features, localization to the epigastrium, relationship to food and periodicity.

Ulcer pain is typically referred to the epigastrium it is localised usually in the acid line or to the right. So that the patient can indicate the site with one finger, 'the pointing sign'. Occasionally ulcer pain is not clearly localised, it may be referred diffusely in the epigastricum, the lower chest or to the back in the interscapular region in the fifth to eighth thoracic segments.

Pain referred to the inter scapular area suggests duodenal or post bulbar ulceration. The description of the pain is not especially helpful, although patients commonly describe it as gnawing or burning.

Most patients recognized a relationship of the pair to the food, although the relationship varies between patients, and in the same patient from time to time. Duodenal ulcer pain tends to occur between meal times, so that the patient may describe it as 'hunger' pain, which is characteristically relieved by food. A notable feature of duodenal ulcer is pain awakening the patient from sleep 2 to 3 hours after refising. The pain of gastric ulcer occurs less regularly; it frequently occurs within an hour of eating, is less often relieved by food and it rarely occurs at night. Besides the characteristic relief obtained after eating, ulcer pain is almost invariably relieved by antacids or by vomiting.

Ulcer pain is characteristically episodic occurring regularly then disappearing to recover ulcers or months later. Between attacks, the patient feels perfectly well, and may eat and drink with impunity. Bouts of pain may at first last only a day os so at a time, and occur only once or twice a year. As the natural history evolves, however episodes begin to last longer and occur more frequently, so that in severe cases remissions of pain may be short lived and pain or discomfort becomes more or less persistent. The cause for these relapses is difficult to be established seasonal factors may be operative, sometimes psychological stress may be blamed, sometimes, dietary

indiscretion and sometimes alcohol in excess. Most commonly no reason can be found for the relapse.

Pain is sometimes absent or so slight as to be described by the patient. Such individuals may complain of other symptoms such as a feeling of "Distension" in the epigastrium or a poorly defined sense of unease after eating. Other complaints include episodic nausea and sometimes anorexia, as well as heartburn or water brash vomiting in clear patients almost always relieves pain and when it is persistent may result in weight loss. This helps to distinguish it from vomiting of psychological origin, in which weight is usually maintained. Persistent vomiting in an ulcer subject usually indicates some degree of gastric narrowing. In such patients, vomiting is usually copious, so that the patient is "surprised" at the volume, the patient often recognizes food ate twelve on more hours previously. Although there is no constant change in bowel rhythm during an ulcer relapse, some patient are aware of constipation or diarrhea when dyspepsia reappears.

Physical Signs

The only physical sign that may be present is 'the pointing sign' which, when accompanied by localized tenderness, is practically diagnostic of an ulcer. However, tenderness may be completely absent, in patients with gastric outlet obstruction, the stomach may be visibly distended, a succession splash may be present and gastric peristalsis may be seen.

Effect of Diet, Drugs, Tobacco and Alcohol

There is no evidence that dietary manipulation affects the symptoms on ulcer healing and therefore strict diets should be avoided. It is particularly important that aspirin and other anti inflammatory drugs are not used in peptic ulceration. Their injurious action should be explained to the patient. There is on evidence that shopping smoking accelerates the healing of gastric ulcers and it is likely to be applied to duodenal ulcers patients should therefore be advised to give up smoking exacerbations of ulcer disease because it aggravates their symptoms. It seems reasonable to encourage moderation in drinking habits in all patients with peptic ulceration.

Surgical Treatment

This has much to offer the patient with intractable peptic ulceration. It can relieve severe or persistent symptoms and prevent complications. While in many patients the assessment of the disability is straight forward, in those in whom anxiety or depression in present, the decision becomes difficult. Elective surgery should be considered in the following circumstances. When the ulcer relapses after several courses cimetidine or ranitidine, or more rarely when the ulcer fails to heal at all, particularly when symptoms, interfere with the enjoyment of life of reduce the capacity to work. The indications for surgery are strengthened if the ulcer has developed in adolescence or young adult life if there is a strong family history or if there has been previous complication such as

hemorrhage or perforation. Finally, some patients fail to comply with medical therapy or express reservations about prolonged therapy. Both points being in favour of elective surgical treatment.

Complications

Complications of peptic ulcer are hemorrhage perforation and gastric out let obstruction and ulcer cancer.

Gastric duodenal Hemorrhage

Gastro duodenal hemorrhage is recognized by hacmatenesis (vomiting of blood) and or melaena (passage of blood in the stools) and usually there are symptoms of hypovolaemia. Upper gastro intestinal haemorrhage carries a mortality that may reach 30% in elderly and shocked patients. A history of significant blood loss within the previous 48 hours should lead to immediate admission to hospital.

Acute Perforation of a Peptic Ulcer:

When free perforation occurs, the contents of the stomach escape into the peritoneal cavity. It perforation occurs without loss of contents as in the accidental perforation of the empty stomach at gastroscopy, few symptoms are produced and the accident may even pass unnoticed. It follows that the symptoms of perforation are those of peritonitis, and they are in proportion to the extent of peritoneal soiling. Occasionally the symptoms of perforation appears and rapidly subside, pre sumably the perforation has then closed spontaneously, or more commonly the ulcer has perforated locally into an area confined by adhesions to adjacent structures. Pertoration occurs more commonly in duodenal than in gastric ulcers and usually in ulcers on the anterior wall. About one quarter of all perforations occur in acute ulcers.

Acute perforation carries a mortality of about 5%. The outlook is poorest in elderly patients. When a large perforation results in extensive peritonitis on when operation is delayed.

Gastric Outlet obstructions:

An ulcer in the region of the pylorus may result in gastric outlet obstruction. This may be due to fibrous structure or to oedema or spasm produced by the ulcer, frequently it is a combination of all three, Long-standing obstruction may lead to severe. 'Retention gastritis' on even the secondary gastric ulcer.

In addition to chronic duodenal ulcer, or benign gastric ulcer at or near the pylorus, gastric outlet obstruction may be caused by carcinoma of the antrum and by a rare condition known as adult hypertrophic pyloric stenosis.

The syndrome of gastric outlet obstruction is loosely described as 'pyloric stenosis'. Even when the cause is chronic duodenal ulcer, and the stenosis is distal to the pylorus thus in 'Pyloric' obstruction due to duodenal stenosis, the pylorus itself may be seen radiologically to be greatly dilated.

Zollinger – Ellison Syndrome:

This is a rare disorder in which severe peptic ulceration occurs due usually to an adenoma or hyperplasia of the islets of the pancreas secreting large amount of gastrin which stimulates the parietal cells of the stomach excessively. The acid output may be so great that the 'acid tide' may reach the upper small intestine, reducing the luminal PH to 2 og less at the pH pancreatic lipase is inactivated and bile acids may be precipitated, causing diarrhea and steaforrhoea . Excessive gastric secretion results in large volumes on aspiration under 'basal' conditions. Pentagastrin does not increase The secretary rate much above 'basal', values, since the stomach is already continuously secreting at or near nominal rates.

Clinical Features:

The ulcers are often multiple and severe and may occur in unusual sites such as the jejunan or the oesophagus. The history is usually short and bleeding and perforation are common. The syndrome may present form of severe recrurrent ulceration following a standard operation for peptic ulcer, the underlying cause not having been recognized.

The diagnosis should be suspected in all patients with unusual or severe peptic ulceration especially coarse barium meal examination shows abnormally coarse gastric mucosal folds. It may be confirmed by finding very high levels of gastrin in the circulation.

DIFFERENTIAL DIAGNOSIS:

1. **Chronic intestinal Amoebiasis:**

   History of recurrent dysentery, caecum and pelvic color are tender and cord like, liver may be palpable and tender, stool may show cysts of entamoeba histolytica.

2. **Chronic cholcystitis:**

   There may be history of biliary colic and jaundice in the past, murphy's sign is positive. Rarely gull bladder may be palpable. Chlocytography settles the diagnosis by showing dysfunction of the gall bladder with or without stone.

3. **Chronic Appendicitis:**

   There may be history of acute appendicitis in the past, Mcburney's point is tender, F.T.M. and barium meal x-ray of appendix may show irregularity or no filling.

4. **Chronic Gastritis:**

   There is anorexia, discomfort in the upper abdomen without any definite tenderness, F.T.M. shows low acid but excess of mucous in all samples, barium meal x-ray shows coarse or tine gastric rugae.

5. Chronic Pancreatitis:

There may be history of acute pancreatitis in the past, pain radiating to the back may be present without definite relation with food. Steatorrhoea and diabetes mellitus may be present, X – ray of the abdomen may reveal pancreatic calcification.

SPECIAL INVESTIGATIONS:

1. Endoscopy in Gastro – Entrology:

In recent years endoscopic photography, both still and motion, has become possible and gives excellent pictures. The flexible fibroscope now enables one to examine the oesophagus, stomach and duodenum and at the same time obtain biopsies and material for cytological examination.

It is used in diagnosis purpose for the oesophagitis, oesophageal ulcer, gastric ulcer, duodenal ulcer, duodenitis malignant cancer, biopsy can also be obtained to find out in gastric ulcer is benign or malignant.

2. Fractional Test Meal:

The patient who was on starvation during the previous night is asked to swallow the ryles tube at 5 a.m. and the entire stomach contents a fasting juice are aspirated with a donel, record syringe. The patient is them given a pint of warm gruel to drink the gruel is prepared by boiling two table spoonfuls of the oatiomeal in two pints of water until the quantity is reduced to one pint. Every 15 minutes not more than 15 ml of gastric contents is now aspirated until 2 ½ hours have elapsed or until such time as 15ml can no longer be aspirated. These samples are examined for total acidity, free Hcl, bile, blood, mucous and starch and the results recorded on a chart In a gastric ulcer, the cures of tree hel, and total acidity are high, normal or just above the normal limit. Blood may be present in some of the specimen. The climbing curve is due to pylorospasm which prevents regurgitation of bile or allows the acidity to rise continuously. Besides carcinoma achlorhydria is found in pernicious anaencia, gastritis, chronic appendicitis, etc, but association or blood in all the specimen is strongly suggestive of a carcinoma. Sometimes cancer cells can be demonstrated into washing after gastric lavage.

This test is no more needed to make correct diagnosis of peptic ulcer except to exclude the role of vagatomy during surgical management.

3. Examination of stool:

Black and farry stool melacna is well known in a peptic ulcer when the hemorrhage is large. Small hemorrhage need special chemical test for detection.

4. Elisa test for H-pylori

The Pyloriset EIA-A III is an enzyme immunoassay for the detection and measurement of H. pylorispecificIgA antibodies in human serum as an aidin diagnosing infection. The product is used to testpatients with symptoms of gastrointestinal disordersand as a follow-up to medical treatment In the assay, the serum samples are added to the microtiter wells coated with specific H. pylori antigens. If anti-H. pylori IgA is present in the specimen, it will bind to the antigen bound to the surface of the wells. The residual test specimen is washed away, and horseradish peroxidase-conjugated anti-human IgA reagent is added. Enzyme conjugate binds to an antigen-antibody complex, unbound conjugate is washed away, and a substrate is added. After stopping the substrate reaction, the color is measured using a photometer. The intensity of the Color
is proportional to the concentration of the H pylori-specific antibody in the sample .

Materials and Methods

MATERIALS AND METHODS

In my clinical study among 40 patients, 20 cases of "Eri Gunam" patients were admitted in postgraduate department of Maruthuvam, Govt. Siddha Medical College Hospital, in-patient ward, and 20 cases were treated in the hospital out-patient department, Chennai-106.

These patients were subjected to a careful examination and the diagnosis was made by Siddha Clinical Methodology and Modern methods.

Patients were selected based on the following signs and symptoms: (inclusion criteria)

- Duration of the illness not more than 10 years.
- Epigastric pain with relation to food.
- Abdominal Discomfort
- Diarrhoea
- Weight loss
- Patients belonging to age group of 20 – 80 years.

Patients were exhausted for the following Criteria: (exclusion criteria)

- ✓ Duration of illness more than 10 years
- ✓ Pyloric stenosis
- ✓ Malignancy in the stomach and in the other parts of the body.
- ✓ Acute abdominal colics
- ✓ Pain due to gallbladder stone (or) pancreatitis.
- ✓ Patients belonging to age groups below 20 and above 80 years.

Patients from both sexes of various age groups were admitted based on the above criteria. First priority was given to clinical findings and confirmatory diagnosis was made by conducting all the necessary investigation in Siddha as well as modern aspects. Hence from these signs and symptoms of Eri Gunmam correlates with the peptic ulcer in modern system of medicine investigations, which meant for peptic ulcer in modern medicine is carried out endoscopy for Eri Gunmam also.

Siddha diagnosis was made with the help of the following criteria:

Regarding Siddha System:

1. Thinaigal (Ivagai Nilai)
2. Paruva Kalangal
3. Mukkutra Nilaigal
4. Elu Udal Kattugal
5. Envagai Thervugal

In Modern system of Medicine the following investigation were done:

Routine Laboratory Investigations:

1. Haemotological investigations:

- ❖ Total WBC count
- ❖ Differential WBC Count
- ❖ Erythroucyte sedimentation rate (ESR)
- ❖ Haemoglobin percentage (Hb%)
- ❖ Blood Sugar, Urea
- ❖ Blood Groups
- ❖ Serum cholesterol.

2. Urine Analysis

- ❖ Albumin
- ❖ Sugar
- ❖ Deposits

3. Stools Examination

- ❖ Ova
- ❖ Cyst
- ❖ Occult blood

4. Special Investigation

- ❖ Endoscopy
- ❖ Serum Amylase
- ❖ USG Abdomen

Selection of medicines:

Selection of medicines was made after in depth study of various siddha literatures.

The test medicines are

1. **Gunmathi Choornam** – 1 g 3 times / day with Hot water / milk, After Food.

2. **Musumusukai Lehyam** - 5 gm 2 times / day after food.

The biochemical constituents and microbiological effects of the drug were studied. The drug was also subjected to the pharmacological and toxicological tests in Albino rat modes.

Results and Observation

RESULTS AND OBSERVATIONS

For this study 40 cases were selected. all these cases clinical examinations under both Siddha and modern aspects were observed and results were taken. Results were observed with respects to the following criteria.

- ✓ Sex distribution
- ✓ Age distribution
- ✓ Socio-Economic Status
- ✓ Duration of illness
- ✓ Dietary habits
- ✓ Nilam (Thinai)
- ✓ Paruva Kaalam
- ✓ Kaalam
- ✓ Religion Reference
- ✓ Distribution of Vatham, pitham, kabham
- ✓ Udal Kattugal
- ✓ Envagai Thervugal
- ✓ Naadi
- ✓ Blood Grouping
- ✓ Clinical Features
- ✓ Results.

All the patients were treated with medicines.

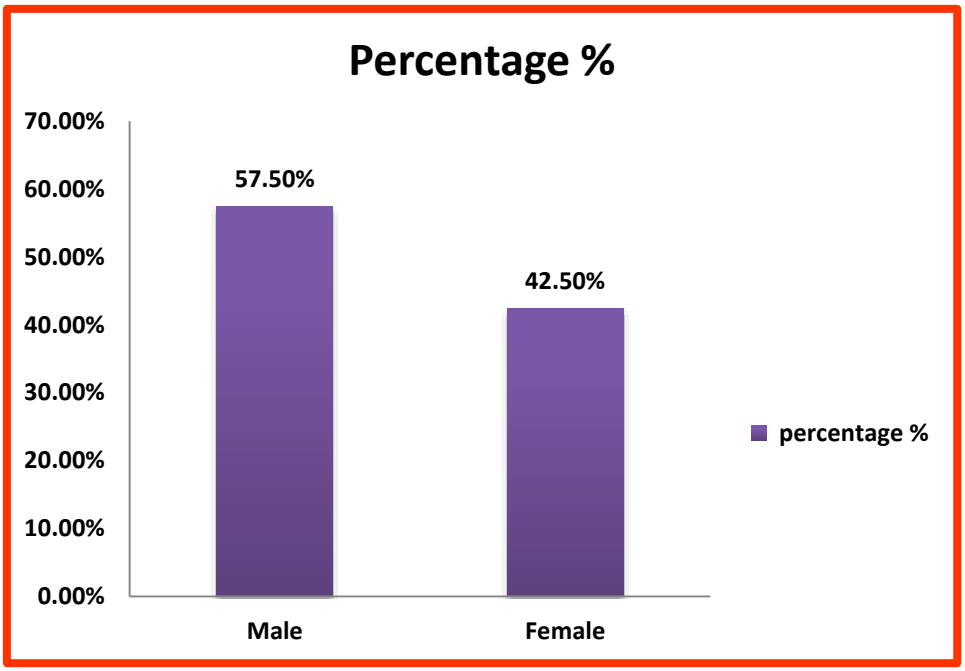
1. **Gunmathi Choornam** – 1 g 3 times / day with Hot water / milk, After Food.

2. **Musumusukai Lehyam -** 5 gm 2 times / day after food.

Among the 40 cases the epigastric pain, burning sensation, abdominal discomfort, diarrhoea, and weight loss and other symptoms were relieved after the administration of the medicines Digestion with good appetite and no chronic effects were observed.

1. SEX DISTRIBUTION

S.No.	Sex	No. of cases	Percentage %
1.	Male	23	57.5%
2.	Female	17	42.5%

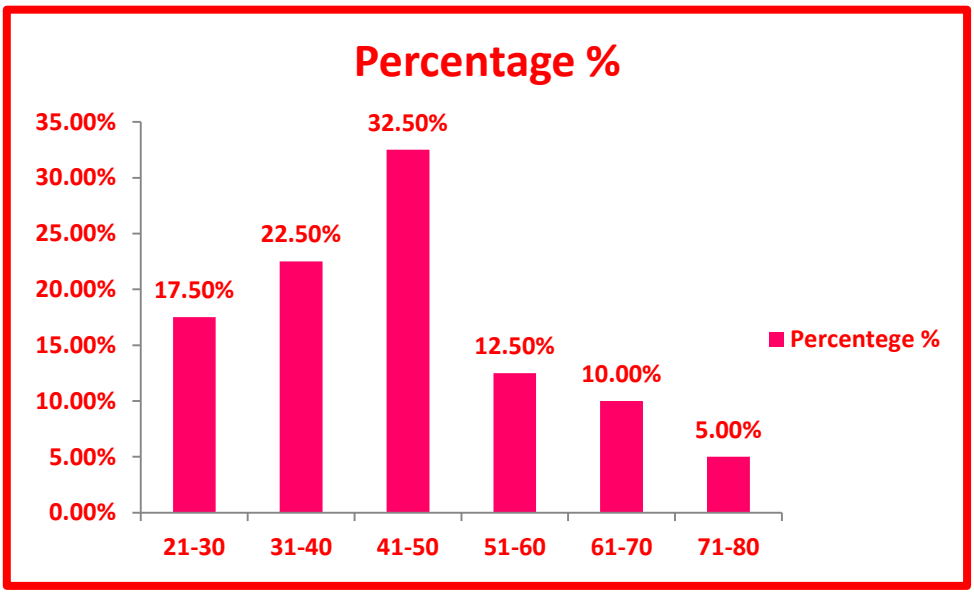


Inference:

Out of 40 patients 23 cases (57.5%) were male and 17 cases (42.5%) were female.

2. AGE DISTRIBUTION

S.no	Age	No of cases	Percentage %
1	21-30	7	17.5%
2	31-40	9	22.5%
3	41-50	13	32.5%
4	51-60	5	12.5%
5	61-70	4	10.0%
6	71-80	2	5.0%

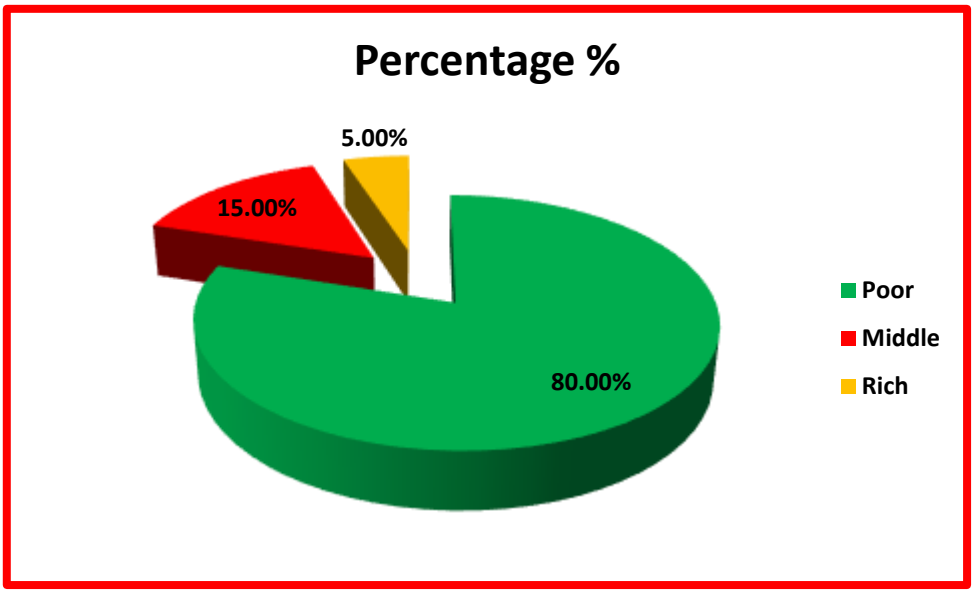


Inference:

Among 40 patients, 7 belong to age group 21 – 30 and 9 patients belong to age group 31 – 40, 13 patients belong to age group 41 – 50, 5 patients belong to age group 51 – 60, 4 patients belong to age group 61 – 70. And 2 patient under 71-80 age group. Maximum patients found between **age group of 41 – 50.**

3. SOCIO ECONOMIC STATUS

S.No.	Socio Economic Status with income group	No. of cases	Percentage%
1.	Poor (Below 5000)	32	80.0%
2.	Middle (5001 to 10000)	6	15.0%
3.	Rich (Above 10000)	2	5.0%

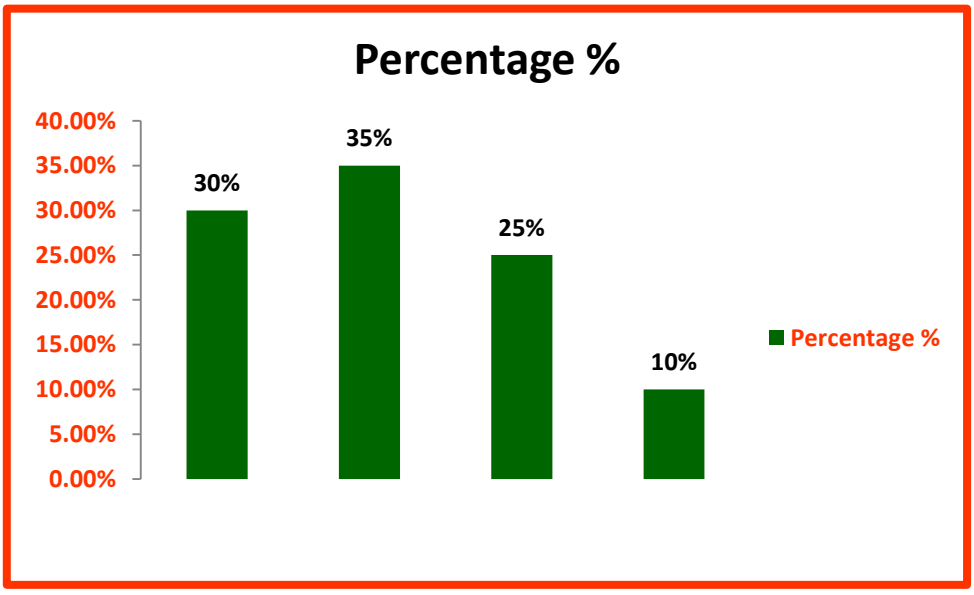


Inference:

Among 40 patients, 32 (80%) were poor, 6 (15%) were middle class and 3 (5%) were rich.

4. DURATION OF ILLNESS

S.No.	Duration	No. of cases	Percentage %
1.	1 month to 6 months	12	30%
2.	6 months to 1 year	14	35%
3.	1 year to 3 years	10	25%
4.	3 years to 5 years	4	10%
5.	5 years and above	-	-

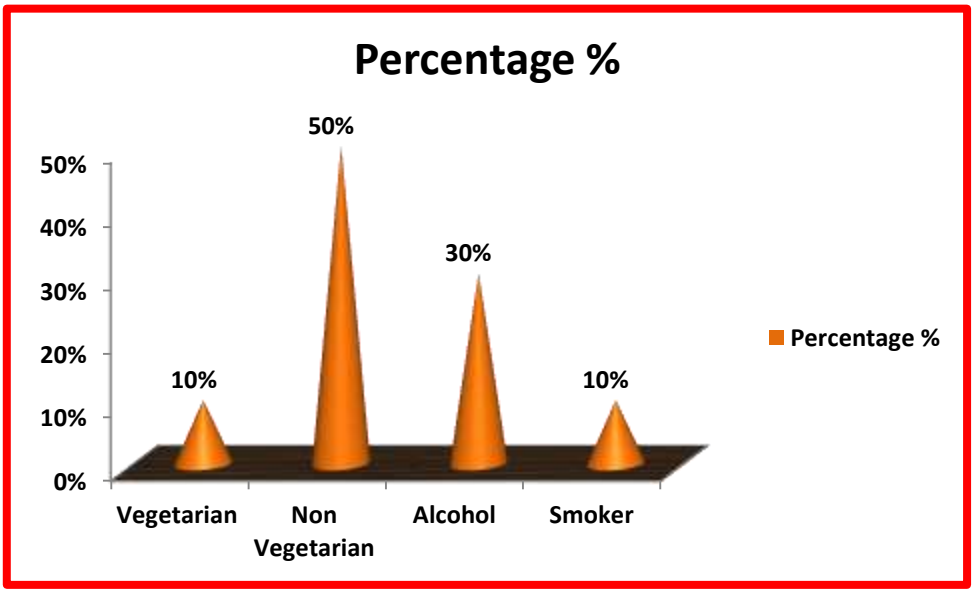


Inference

Most of the cases selected duration of illness is **6 months to 1 year.**

5. HABITS WISH DISTRIBUTION

S.No.	Habits	No. of cases	Percentage%
1.	Vegetarian	4	10%
2.	Non Vegetarian	20	50%
3.	Alcohol	12	30%
4.	Smoker	4	10%

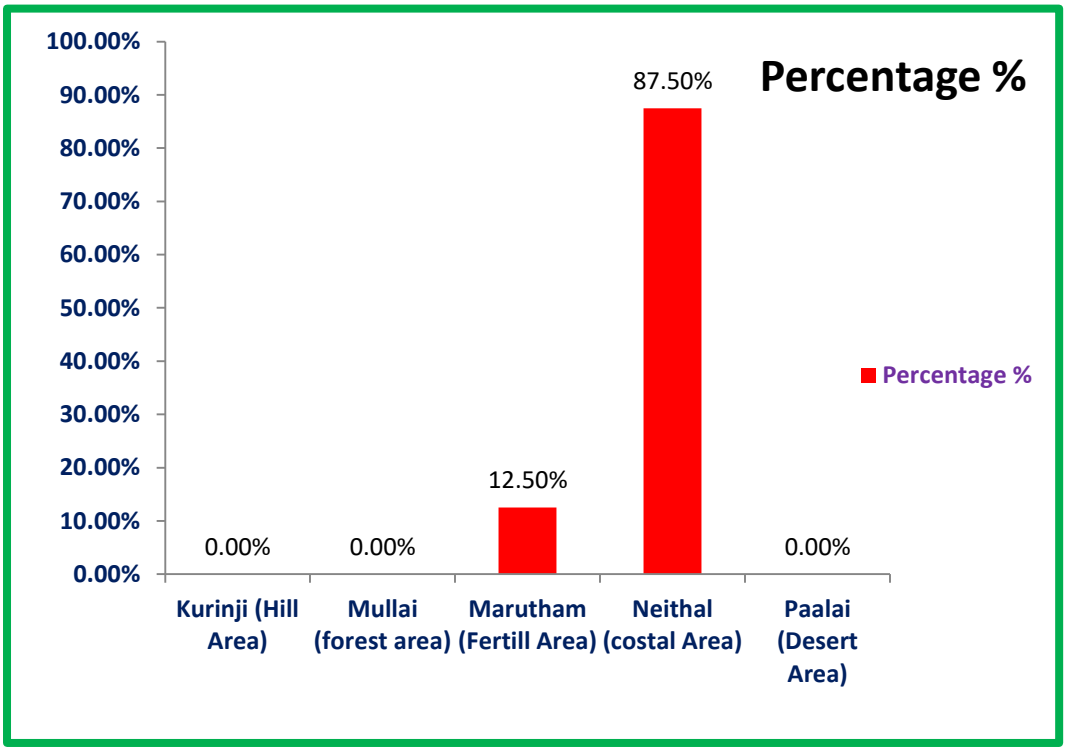


Inference:

Among 40 patients, 4 (10%) were Vegetarian, 20 (50%) were Non-Veg and 12 (30%) were alcoholic, 4(10%) were smoker.

6. NILAM / THINAI

S.No.	Duration	No. of cases	Percentage %
1.	Kurinji (Hill Area)	0	0%
2.	Mullai (forest area)	0	0%
3.	Marutham (Fertill Area)	5	12.5 %
4.	Neithal (costal Area)	35	87.5 %
5.	Paalai (Desert Area)	0	0%

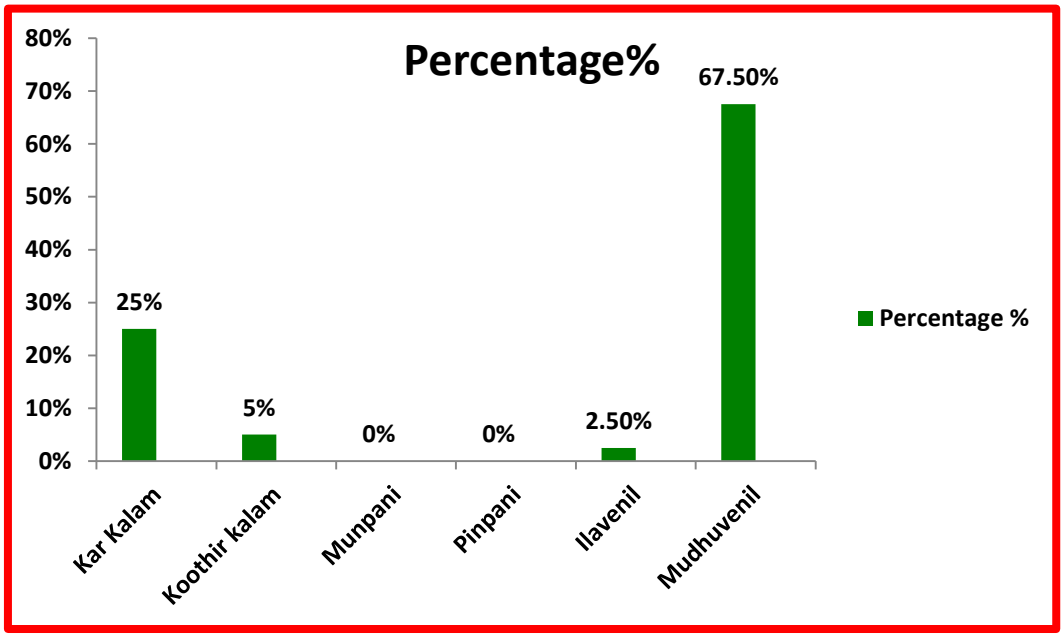


Inference:

Among 40 cases, 35 were belonged to Neithal Nilam, and 5 were belonged to Marudham .

7. PARUVA KALAM

S.No.	Paruva Kalam	Months	No. of cases	Percentage%
1.	Kar Kalam	Avani- Puaratasi (Aug 15 – Oct 15)	10	25.0%
2.	Koothir kalam	Iypasi –Karthigai (Oct 15 – Dec 15)	2	5.0%
3.	Munpani	Margazhi-Thai (Dec'5 – Feb'5)	0	0%
4.	Pinpani	Nasi – Panguni (Feb 15 – April 15)	0	0%
5.	Ilavenil	Chithirai –Vaikasi (April 05-June 15)	1	2.5%
6.	Mudhuvenil	Anni- Aadi (June 15 – Aug 15)	27	67.5%

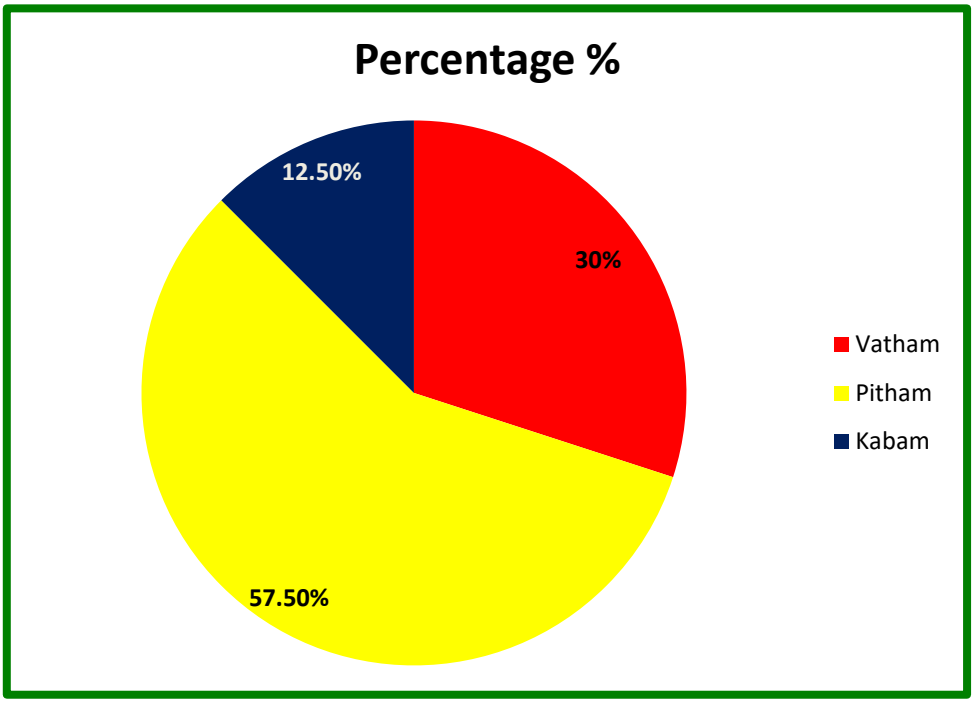


Inference

Among 40 cases 27 cases were belonged to Mudhuvenil Kaalam, 10 cases under Kar kalam,2 cases belonged to Koothir kaalam and 1 were belonged to Ilavenil Kaalam,

8. KAALAM

S.No.	Kaalam	No. of cases	Percentage%
1.	Vatham	12	30%
2.	Pitham	23	57.5%
3.	Kabam	5	12.5%

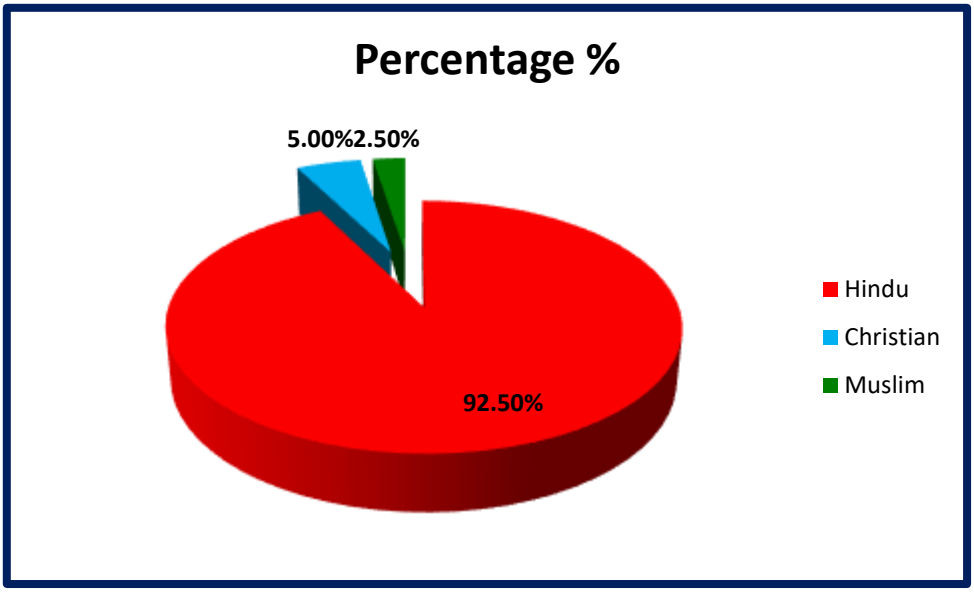


Inference:

Vatha Kaalam lies up to 33rd age of a person, Pitha Kaalam lies between 33rd to 66th age, Kabba kaalam lies above this age. The maximum no of patients of ERI Gunam cases were in Pitha Kaalam

9. RELIGION REFERENCE

S.No.	Religion	No. of cases	Percentage%
1.	Hindu	37	92.5%
2.	Christian	2	5.0%
3.	Muslim	1	2.5 %



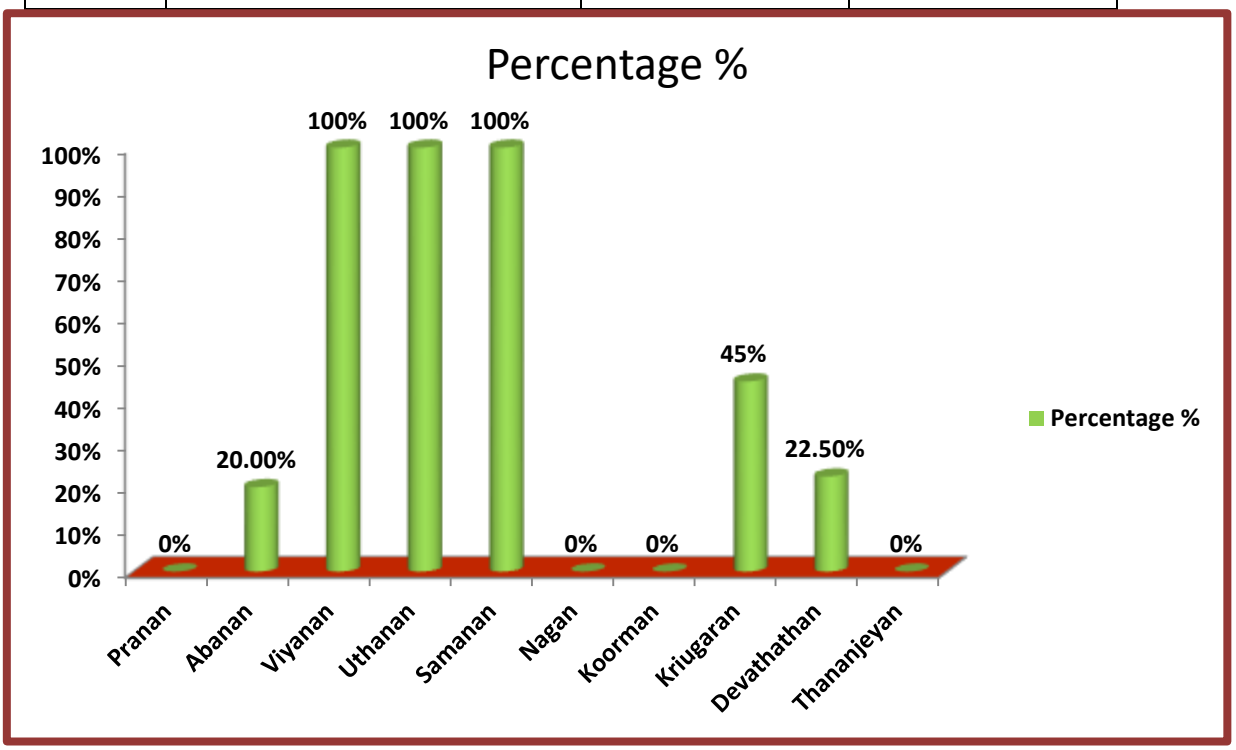
Inference:

Most of the patients religion Hindu 92.50% and Muslims 2.50 %,Christian 5.00 %.

10. DISTRIBUTION OF THRI-DHOSAM

The derangements of the three vital phenomena in 40 cases were studied.

S.No .	Vatham	No. of cases	Percentage%
1.	Pranan	0	-
2.	Abanan	8	20%
3.	Viyanan	40	100%
4.	Uthanan	40	100%
5.	Samanan	40	100%
6.	Nagan	0	-
7.	Koorman	0	-
8.	Kriugaran	18	45%
9.	Devathathan	9	22.5%
10.	Thananjeyan	-	-

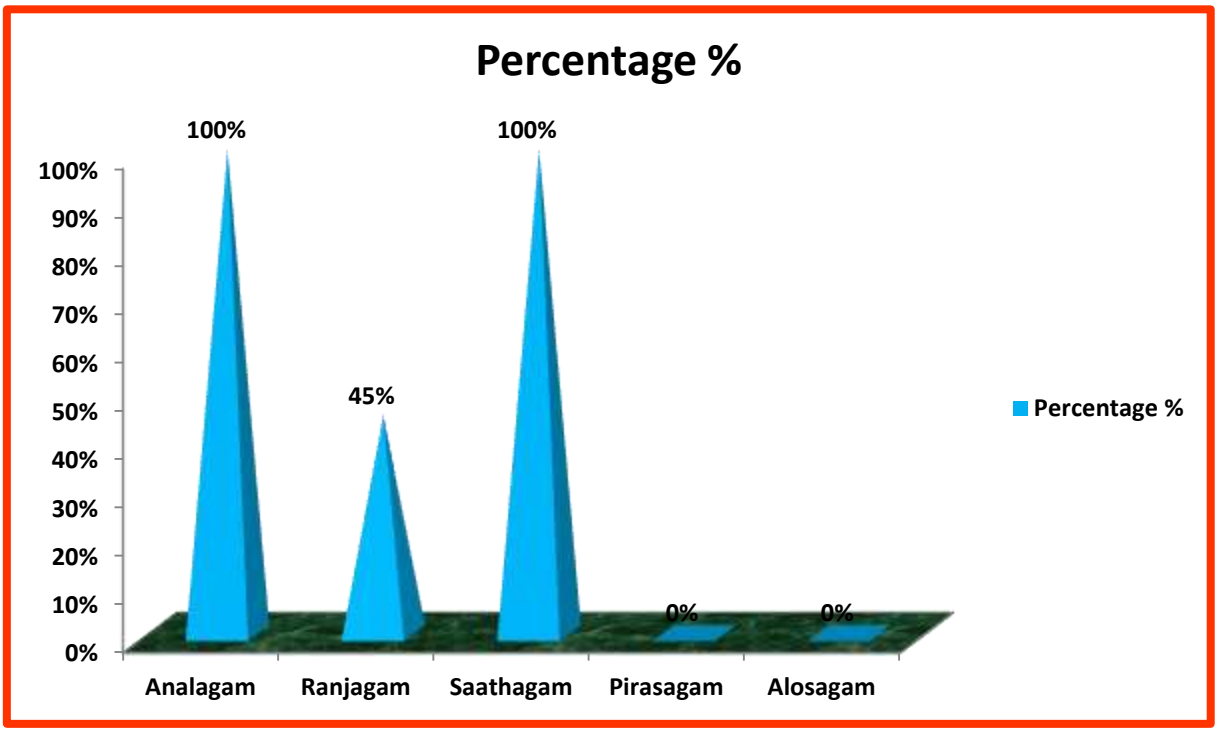


Inference:

Vatha derangement was noted with the disturbance of 10 vaayus, Viyaanan, Uthanan, Samanan and Abanan were the 4 vaayus, mostly affected.

11. PITHAM

S.No.	Pitham	No. of cases	Percentage%
1.	Analagam	40	100%
2.	Ranjagam	18	45%
3.	Saathagam	40	100%
4.	Pirasagam	0	-
5.	Alosagam	0	-

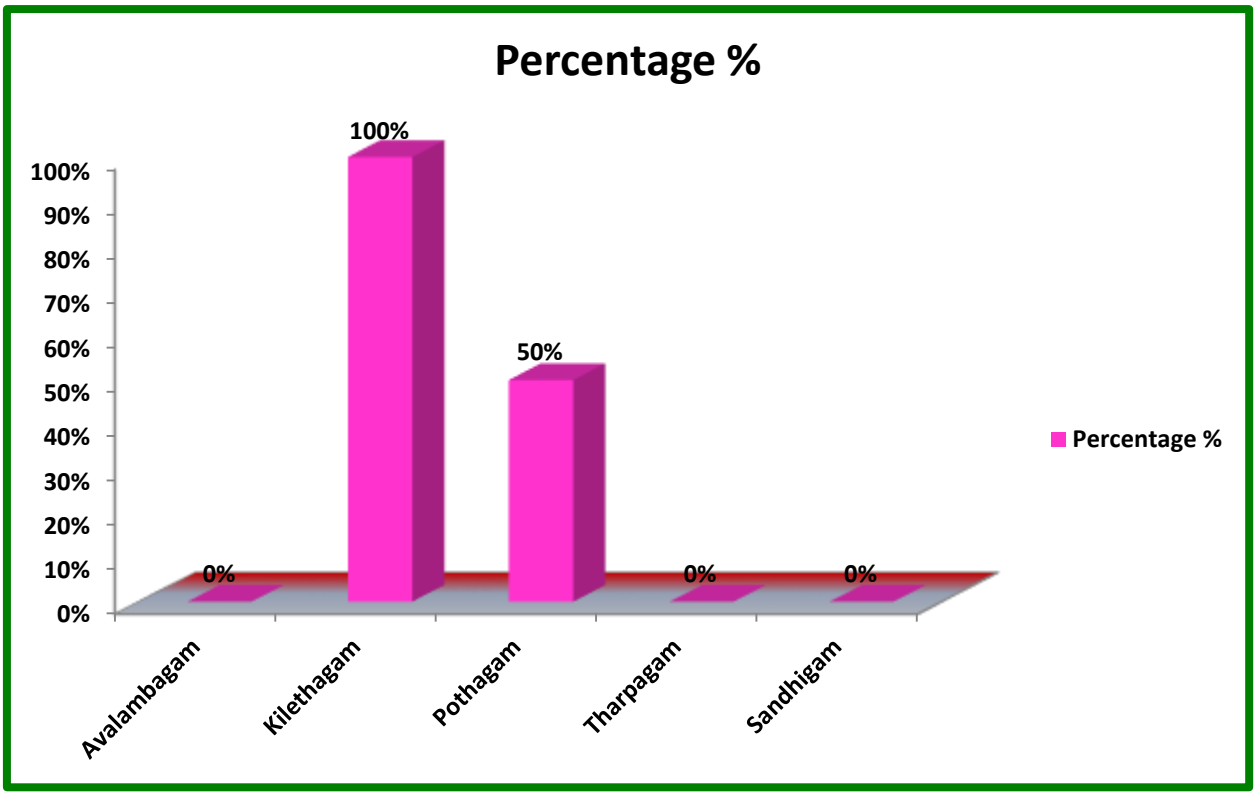


Inference:

In the above five pithas Analagam and Saathagam were mostly affected in the 40 cases.

12. KABAM

S.No.	Kabam	No. of cases	Percentage%
1.	Avalambagam	0	0%
2.	Kilethagam	40	100%
3.	Pothagam	20	50%
4.	Tharpagam	0	0%
5.	Sandhigam	0	0%

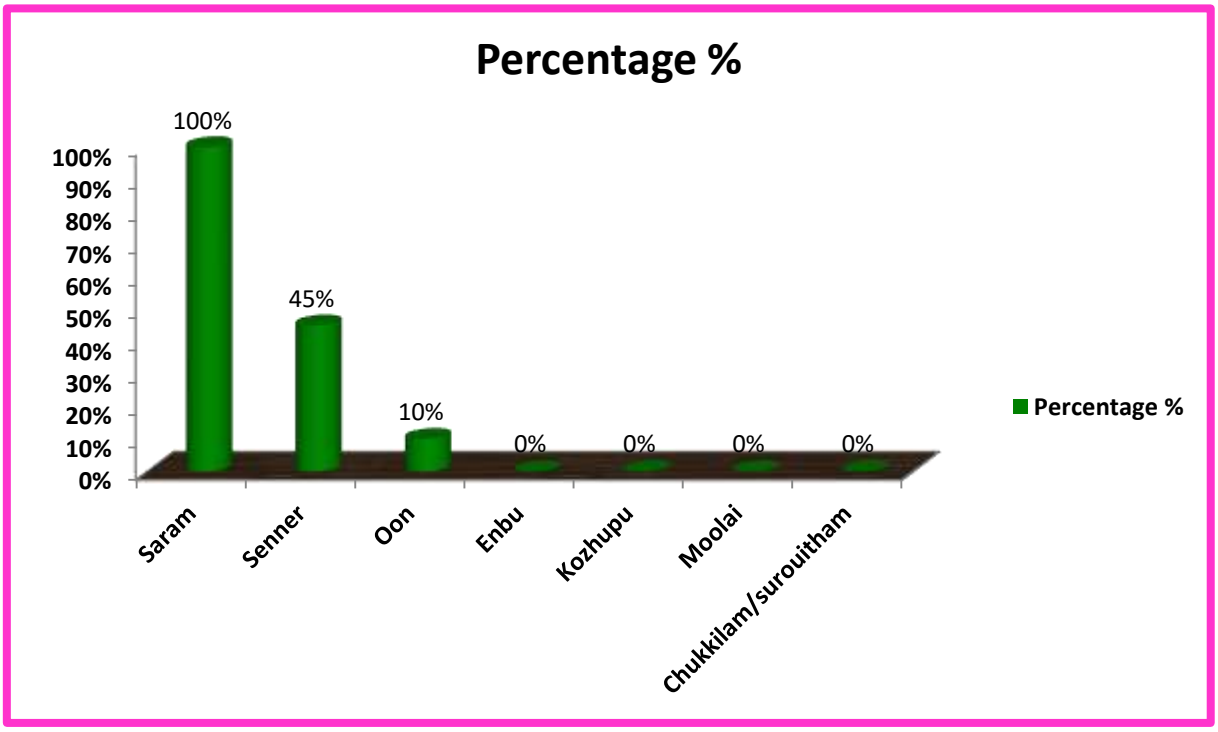


Inference:

From this study, it is seen that in all the cases Kilethagam was chiefly affected in100% and in 50% of the cases pothagam was affected.

13. UDAL KATTUGAL

S.No.	Udal Kattugal	No. of cases	Percentage%
1.	Saram	40	100%
2.	Senner	18	45%
3.	Oon	4	10%
4.	Enbu	0	0%
5.	Kozhupu	0	0%
6.	Moolai	0	0%
7.	Chukkilam/surouitham	0	0%

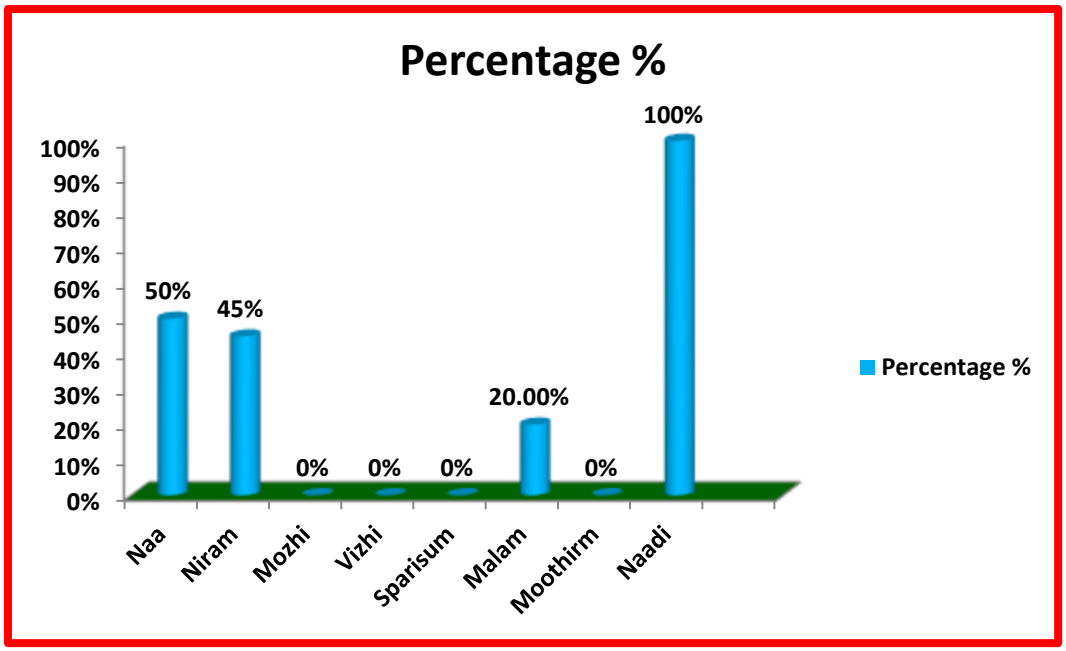


Inference:

In all the cases, Saaram was chiefly affected in 100% of the cases, seneer had been affected in 45% of the cases, Oon was affected in 10% of the cases.

14. ENVAGAI THERVUGAL

S.No.	Envagai thervugal	No. of cases	Percentage%
1.	Naa	20	50%
2.	Niram	18	45%
3.	Mozhi	0	0%
4.	Vizhi	0	0%
5.	Sparisum	0	0%
6.	Malam	8	20%
7.	Moothir	0	0%
8.	Naadi	40	100%

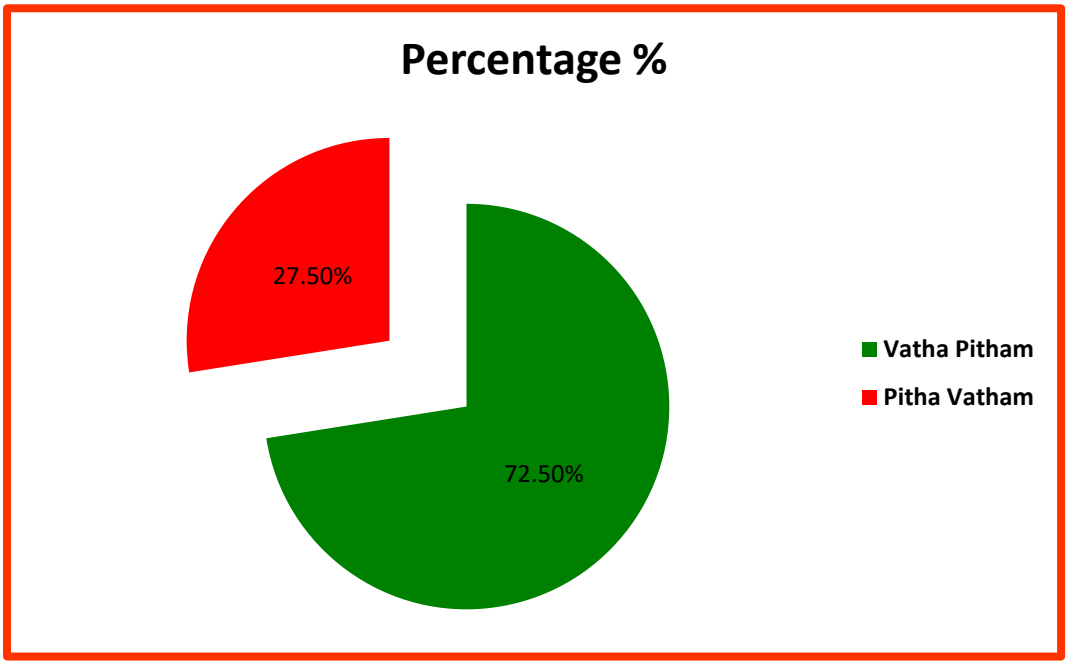


Inference:

After the confirmation of diagnosis as Gunmam, the type of Gunmama is confirmed by comparing the identies and differences of signs and symptoms and results obtained by Envagai Thervugal.

15. NAADI

S.No.	Naadi	No. of cases	Percentage%
1.	Vatha Pitham	29	72.5%
2.	Pitha Vatham	11	27.5%

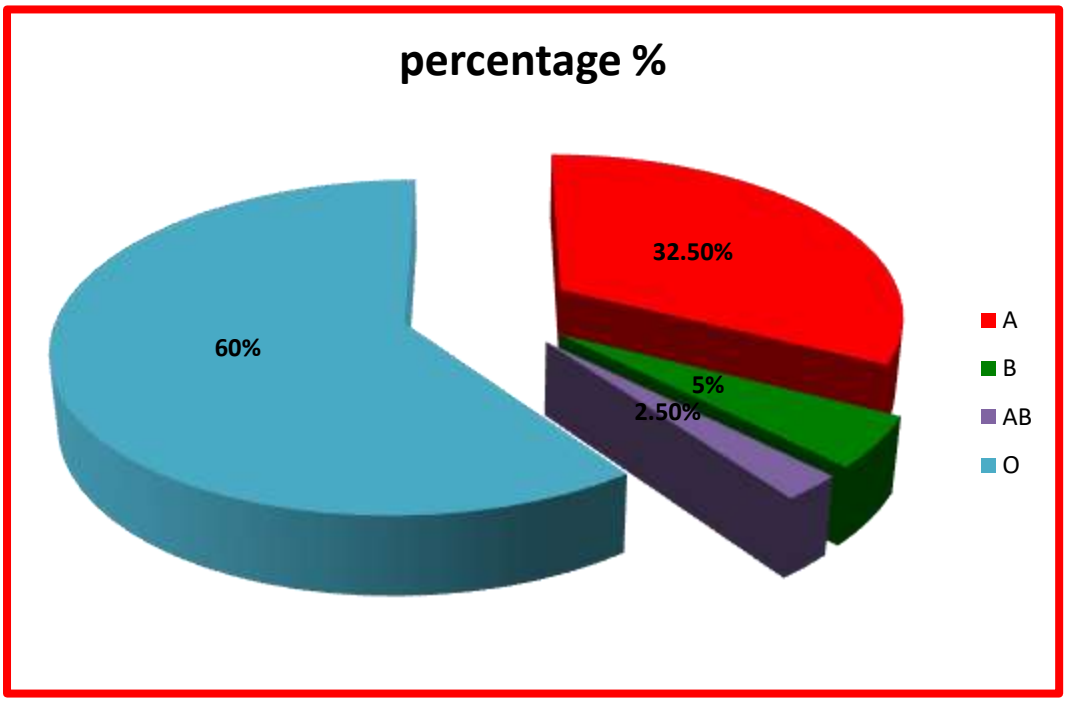


Inference:

Among 40 cases 72.50% patients have Vatha pitham and 27.50% Pitha Vatham.

16. BLOOD GROUPING

S.No.	Blood Grouping	No. of cases	Percentage%
1.	A	13	32.5%
2.	B	2	5%
3.	AB	1	2.5%
4.	O	24	60%

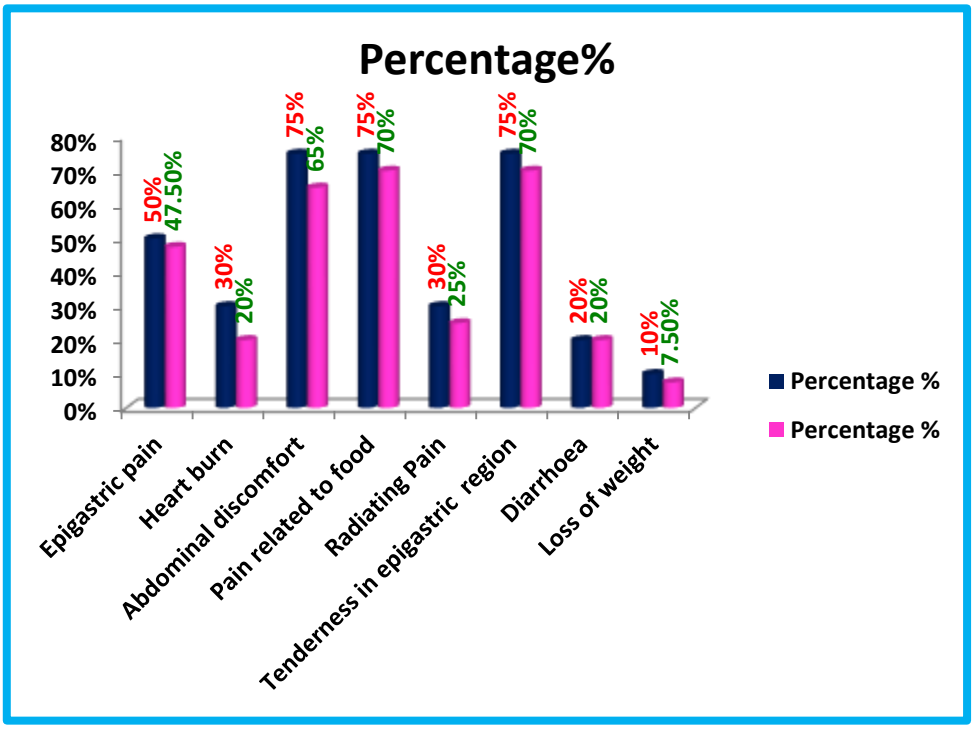


Inference:

From this study it is seen that maximum incidence of the disease Eri Gunmam seen in the patients belonged to "**O**" Group.

17. CLINICAL FEATURES

		No. of patients affect		No. of patients improved	
S.No.	Symptoms	Before treatment	Percentage %	After treatment	Percentage %
1.	Epigastric pain	20	50%	19	47.5%
2.	Heart burn	12	30%	8	20%
3.	Abdominal discomfort	30	75%	26	65%
4.	Pain related to food	30	75%	28	70%
5.	Radiating Pain	12	30%	10	25%
6.	Tenderness in epigastric region	30	75%	28	70%
7.	Diarrhoea	8	20%	8	20%
8.	Loss of weight	4	10%	3	7.5%



Among 20 patients 19 pts improved from Epigastric Pain

Among 12 patients 8 pts improved from Heart burn

Among 30 patients 26 pts improved from Abdominal discomfort

Among 30 patients 28 pts improved from pain related to food.

Among 12 patients 10 pts improved from radiating pain.

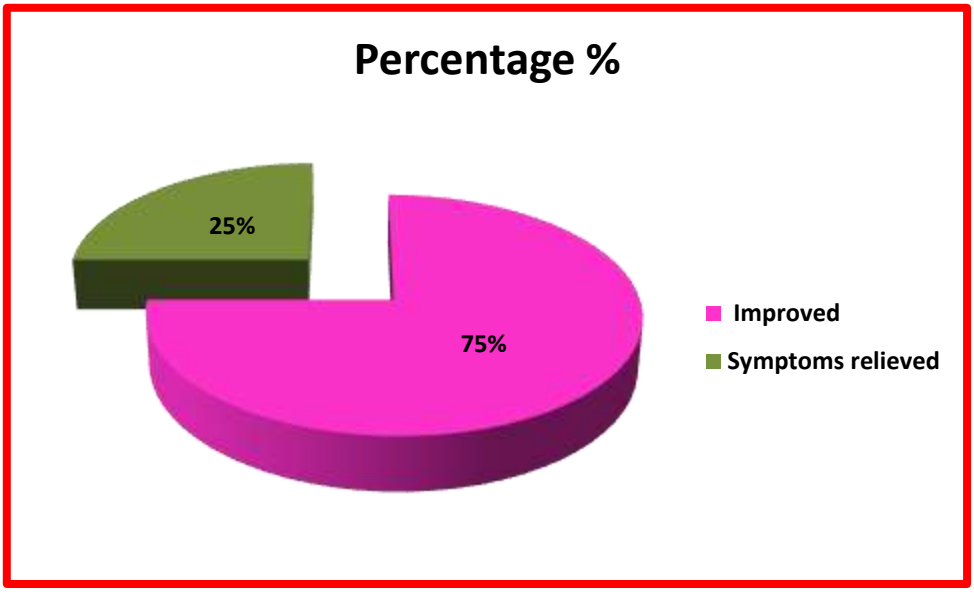
Among 30 patients 28 pts reatigot relief from tenderness in Abdominal region.

Among 8 patients 8 pts got relief from Diarrhoea.

Among 4 patients 3 pts improved from loss of weight.

18. RESULTS

S.No.	Results	No. of cases	Percentage%
1.	Improved	30	75 %
2.	Symptoms relieved	10	25 %



Inference:

Results obtained Were

- 75% of cases Improved.
- 25% of cases Symptoms relieved

The results were based on clinical improvement.

S.No	Name of the Patients	Blood Group	Age/ Sex	B.T	Indication	A.T
1.	Lakshmi	A	35/F	Taken	Gastric Ulcer	Improved
2.	Visvanathan	O	50/M	Not Taken	Gastric ulcer	Symptoms relieved
3.	Gowthmraj	A	25/M	Not Taken	Gastric Ulcer	Symptoms relieved
4.	Tamilarsan	O	30/M	Taken	Duodenal Ulcer	Improved
5.	Susila	A	48/F	Taken	Duodenal ulcer	Improved
6.	Balu	A	42/M	Not Taken	Gastric ulcer	Symptoms relieved
7.	Paul	O	32/M	Taken	Duodenal Ulcer	Improved
8	Nagaraj	A	32/M	Taken	Duodenal Ulcer	Improved
9.	Parthasarathi	O	28/M	Not Taken	Gastric ulcer	Symptoms relieved
10.	Govindraj	O	42/M	Not Taken	Gastric Ulcer	Improved
11.	Ramkumar	O	32/M	Not Taken	Gastric ulcer	Improved
12.	Senthilvel	O	30/M	Not Taken	Gastric ulcer	Improved
13.	Ramu	AB	31/M	Taken	Gastric Ulcer	Improved
14.	Mahendran	O	29/M	Not Taken	Duodenal Ulcer	Improved
15.	Deenadayalan	A	33/M	Not Taken	Duodenal Ulcer	Improved
16.	Rathikumar	O	37/M	Taken	Gastric Ulcer	Improved
17.	Rani	O	44/F	Taken	Gastric Ulcer	Symptoms relieved
18.	Gnnasekaran	A	48/M	Not Taken	Duodenal ulcer	Improved
19.	Karthikayan	A	21/M	Taken	Duodenal Ulcer	Improved
20.	Shakthivel	O	29/M	Not Taken	Duodenal Ulcer	Improved
21.	Lakshmidevi	B	38/F	Not Taken	Gastric Ulcer	Improved
22.	Poongavanam	O	70/F	Not Taken	Gastric ulcer	Symptoms relieved
23.	Thayammal	O	70/F	Not Taken	Duodenal Ulcer	Improved
24.	Amsavalli	O	66/F	Not Taken	Duodenal Ulcer	Improved
25.	Mari	O	50/M	Not Taken	Gastric ulcer	Improved
26.	Deivanai	O	47/F	Not Taken	Gastric ulcer	Improved

27.	Thilagam	O	50/F	Not Taken	Gastric ulcer	Improved
28.	Nagammal	A	60/F	Not Taken	Duodenal ulcer	Symptoms relieved
29.	Palani	O	50/M	Not Taken	Duodenal Ulcer	Improved
30.	Ameer	O	75/M	Not Taken	Gastric ulcer	Improved
31.	Gajendran	O	60/M	Not Taken	Gastric ulcer	Improved
32.	Selvi	O	50/F	Not Taken	Duodenal Ulcer	Symptoms relieved
33.	Marimuthu	A	60/M	Not Taken	Gastric ulcer	Improved
34.	Papathi	O	59/F	Not Taken	Gastric ulcer	Improved
35.	Shankar	B	35/M	Not Taken	Gastric Ulcer	Improved
36.	Dharani	O	72/M	Not Taken	Duodenal Ulcer	Symptoms relieved
37.	Veerappan	A	40/M	Not Taken	Duodenal Ulcer	Improved
38.	Vijalakshmi	O	60/F	Not Taken	Gastric ulcer	Improved
39.	Usha	A	43/F	Not Taken	Duodenal ulcer	Symptoms relieved
40.	Sargunam	O	46/F	Not Taken	Gastric ulcer	Improved

Even though all patients were insisted for endoscopic investigations, only 11 of them was willing ,since others faceing inconveniences during these investigation before treatment.

Other than these endoscopic investigations, rests of the patients are clinically diagnosed by their clinical features, signs and symptoms.

MedIndia Hospitals

(Superspeciality Digestive Diseases & Allied Multi Speciality Institution)

83, Valluvar Kottam High Road (Village Road), Nungambakkam

Chennai - 600 034 ☎ - 91 - 44 - 28311001, 28311415, 28204757.

UPPER G.I. SCOPY REPORT

NAME : Mr.PAUL **MDH No.** : 00859K

AGE : 32 **SEX** : MALE **DATE** : 03/05/2011

SCOPE PASSED UPTO : *Duodenum II part*

PHARYNX : *Normal*

OESOPHAGUS : *Grade B oesophagities.*
LAX LES.

OG - JUNCTION : *Normal*

STOMACH : *Fundus ,normal Body erethimatus & Antrum are normal.*

DUODENUM I PART : *Erosions.*

DUODENUM II PART : Normal

RUT : ~~Negative~~ +ve

IMPRESSION : * *EROSIVE DUEDENETES.WITH*
* *LAX LES.*

Oesophagus

Fundus

Antrum

Duodenum IInd Part

Duodenum Ist part

Dr.V.S. SANKARANARAYANAN MD.

LABORATORY INVESTIGATION REPORTS (OP/IP)

Sl. No.	O.P. No.	Name/SEX	Age	HAEMATOLOGICAL REPORT														URINE ANALYSIS						STOOL ANALYSIS	
				BEFORE TREATMENT				AFTER TREATMENT				ESR (mm)				Hb(Gm)		BT			AT			BT	AT
				TC CU/mm	DC %			TC CU/mm	DC %			BT		AT		BT	AT	Alb	Sug	Dep	Alb	Sug	Dep	OB	OB
					P	L	E		P	L	E	½ hr	1 hr	½ hr	1hr										
1.	573	LAKSHMI/F	35	9000	55	38	7	10500	62	34	4	22	45	15	30	9.0	12.5	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
2.	222	VISVANATAN/M	50	9400	59	36	5	10100	62	34	4	5	12	4	7	11.8	13.5	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
3.	593	TAMILARSAN/M	30	10100	57	30	13	9600	53	39	8	10	22	7	15	9.0	13.4	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
4.	3095	GOWMTHAMRAJ /M	25	9000	57	39	4	9800	60	36	4	5	11	5	10	13.0	15.0	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
5.	932	SUSILA/F	48	10200	62	33	5	10000	52	33	5	15	38	10	22	10.4	12.5	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
6.	7082	BALU/M	42	9400	59	36	5	9800	60	36	4	15	5	11	30	12.4	15.0	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
7.	737	PAUL/M	32	7800	56	38	6	10100	60	36	4	2	7	5	10	8.0	11.5	NIL	NIL	Opc	NIL	NIL	NIL	+	NIL
8.	3497	NAGARAJ/M	32	9800	59	36	5	10200	60	34	6	3	6	5	10	16.0	12.4	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
9.	5510	PARTHASARATHY/M	28	9800	59	37	4	8900	55	41	4	15	20	5	12	13.0	12.0	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
10.	2932	GOVINDRAJA/M	32	10200	60	34	6	10400	60	36	4	5	11	25	60	13.0	12.5	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
11.	6735	RAMKUMAR/M	32	10400	60	34	6	9600	50	45	5	8	25	12	25	13.0	15.0	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
12.	6889	SENTHILVEL/M	30	9100	57	38	5	9800	60	34	6	5	20	15	33	11.2	8.0	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
13.	6729	RAMU/M	31	9700	57	38	5	9200	55	41	4	5	12	5	11	12.0	12.0	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
14.	8886	MAGENDRAN/M	29	9800	57	39	4	9400	57	38	5	12	13	10	18	10.0	13.4	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
15.	8045	DEENADAYALAN /M	33	9700	59	33	8	9700	69	33	8	5	13	8	18	12.6	12.6	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
16.	6684	RATHIKUMAR/M	37	10000	57	38	5	9000	54	42	4	5	12	5	7	16.0	13.0	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
17.	2430	RANI/F	44	9000	53	40	7	10200	61	35	4	9	25	7	12	10.0	12.0	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
18.	1733	GYNASEKAR/M	48	10400	66	28	6	10100	60	36	4	7	16	10	15	12.0	14.5	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
19.	7400	KARTHIKAYAN/M	21	9000	54	42	4	9800	56	41	3	2	8	5	11	12.0	15.0	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
20.	8357	SAKTHIVEL/M	29	9400	55	41	4	9500	61	35	1	2	4	5	10	12.5	14.0	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL

TC – Total Count , Hb – Hemoglobin, Dc – Differential Count , P – Polymorph, L – Lymphocyte , E – Eosinophil , ESR – Erythrocyte Sedimentation Rate, OEC – Occasional Epithelial Cells

OPC – Occasional Pus Cells, FPC – Few Pus Cells, FEC – Few Epithelial Cells , OB - Occult Blood, Alb – Albumin, Sug – Sugar , Dep – Deposits.

Sl. No.	I.P. No.	Name/SEX	Age	HAEMATOLOGICAL REPORT														URINE ANALYSIS						STOOL ANALYSIS	
				BEFORE TREATMENT				AFTER TREATMENT				ESR (mm)				Hb(Gm)		BT			AT			BT	AT
				TC CU/mm	DC %			TC CU/mm	DC %			BT		AT		BT	AT	Alb	Sug	Dep	Alb	Sug	Dep	OB	OB
					P	L	E		P	L	E	½ hr	1 hr	½ hr	1hr										
1.	826/5908	LAKSHMIDEVI/F	38	9200	53	41	6	9000	56	44	4	20	50	15	30	8.7	12.5	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
2.	600/7040	POONGAVANAM/F	70	6100	60	41	5	9000	56	40	3	60	90	10	25	5.0	12.0	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
3.	122/8665	THAYAMMAL/F	70	8700	58	36	6	9200	58	34	3	28	17	12	20	9.5	11.5	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
4.	156/9347	AMSAVALLI/F	66	9500	55	32	4	9000	52	34	3	7	18	10	15	9.0	12.0	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
5.	941/169	MARI/M	50	10600	62	30	8	9200	53	32	4	15	35	10	15	11.5	14.0	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
6.	630/8078	DEIVANAI/F	47	10700	60	36	4	9600	60	34	3	14	40	10	15	9.0	11.5	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
7.	875/7739	THILAGAM/F	50	9000	55	38	4	9000	53	34	3	40	72	20	30	6.0	10.5	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
8.	214/6518	NAGAMMAL/F	65	9000	53	37	8	9200	56	39	3	27	50	12	25	9.0	11.5	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
9.	52/5207	PALANI/M	50	9000	59	38	3	9000	60	40	3	12	39	12	22	11.5	10.5	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
10.	690/1214	AMEER/M	75	9800	57	39	4	9200	63	40	3	10	16	12	22	8.0	12.5	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
11.	816/5632	GAJENDRAN/M	60	9600	59	35	6	9000	60	35	4	55	87	20	32	9.0	11.5	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
12.	789/4600	SELVI/F	55	9400	53	31	4	9000	60	42	4	25	32	12	20	9.5	11.0	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
13.	99/7299	MARIMUTHU/M	60	9700	58	34	2	9200	70	42	2	20	34	12	20	11.0	12.0	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
14.	544/4892	PAPATHI/F	59	9100	57	39	4	9400	57	38	5	12	13	10	18	10.0	13.4	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
15.	545/4907	SHANKAR/M	35	9700	59	43	5	9700	69	33	4	5	13	8	18	12.6	14.0	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
16.	932/9669	DHARANI/M	72	8900	55	39	6	9000	60	40	3	44	85	18	32	9.0	11.0	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
17.	2/363	VEERAPPAN/M	40	10000	54	32	4	10200	62	42	4	10	25	10	20	11.0	13.0	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
18.	549/4971	VIJAYALAKSHMI/F	60	6000	53	41	6	9000	60	40	3	35	80	15	30	5.0	9.0	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL
19.	437/774	USHA/F	43	8600	55	42	4	9000	60	42	3	10	18	10	12	9.2	11.5	NIL	NIL	Opc	NIL	NIL	NIL	NIL	NIL
20.	977/2015	SARGUNAM/F	46	9100	57	38	5	9000	60	42	3	13	27	10	22	8.9	10.0	NIL	NIL	Opc	NIL	NIL	Opc	NIL	NIL

Discussion

DISCUSSION

40 cases of Eri Gunmam attended in the Post graduate department of Maruthuvam, Govt.Siddha Medical College, Chennai-600 106. Out of 40 cases 20 cases which had severe symptoms were selected for admission in the In-patient ward. In addition the remaining 20 cases were treated regularly in the outpatient department for about 30 days.

The patients were examined based on Siddha and as well as modern aspects and all the necessary investigations were made during the history taking. The results obtained from their studies were discussed below.

Age Distribution

Among 40 cases 17.5% of the cases were in the age group from 21 to 30 years, 22.5% of the cases were from 31 to 40, and 32.5% of the patients were from 41 to 50, 12.5 % of the patients were from 51 to 60, 10% of the patients were from 61 to 70 and 5% of the patients were71 to 80.

Sex Distribution

Among 40 cases 23 were male and 17 were female.

Socio Economic Status

Among 40 cases, 80% of the cases were poor (below 5000), 15% of the cases were from middle (5001 to 10000) and 5% of the cases were from high income (above 10000) group.

Duration of the Illness

In 30% of the cases the duration of illness was from 1 month to 6 months, 35% of the cases, the duration was from 6 months to 1 year, 25% of the cases the duration was from 1 year to 3 years, 10% of the cases the duration was from 3 years to 5 years, none of the cases the duration of illness was from 5 years and above.

Clinical Features

In this study it is observed that all the patients taken for this study had a epigastria pain abdominal discomfort, Heartburn, diarrhea, loss of weight as the main clinical features. 75% of them had pain related to food , abdominal discomfort and tenderness in abdominal region. 50% of them had epigastria pain, 30 % of them had heart burn and radiating pain, 20% of them had diarrhea and 10% of them had loss of weight.

Diet and Habits

Among 40 cases, 4(10%) were Vegetarian, 20(50%) were Non-Veg, 4(10%) were smoker 12(30%) were alcoholic.

Nilam Thinai

Among 40 cases 87.5% of them Neithal and 12.5% of them Marutham.

Paruva Kalam

Out of 40 cases studied 67.5% of cases encounted in Mudhuvenil Kalam, 25% cases in Kar kalam,5% cases in Koothir kalam,and2.5% cases in Ilavenil Kalam.

Kalam

Among 40 cases 30% cases were in Vatha Kalam, 57.5% cases were in Pitha Kalam, and12.5% of cases were in Kabha kalam.

Investigation

An the 40 casaes were under routine laboratory investigation at the time of admission and discharge the urine and stool analysis reveals no significant abnormalities.

Blood Group

Among the 40 cases 24 cases were belongs to 'O' blood group, 13 cases were belongs to 'A' group, 2 cases were belongs to 'B' group and 1 case belongs to 'AB' group.

Endoscopy Investigation

Out of the 40 cases 11 cases were put to the Endoscopy study at the laboratories and in that 5 cases were confirmed as duodenal ulcer and the remaining 6 cases were gastric ulcer.

Trial Medicines

The trial medicines was administered to all the cases selected for study.

1. Gunmathi Choornam – 1g, 3 times/day, After meals

2. Musumusukai Lehyam – 5gm-2 times/day, after meals

Both the drugs Gunmathi Choornam and Musumusukai Lehyam produced anti ulcer activity which was found to the significant when compared with the control. The anti ulcer activity of both the drugs were comparable with that of the standard drug Ranitidine which is also significant when compared with the control.

The trial medicines were subjected to the Pharmacological and Bio-Chemical analysis.

The reports of the medicines had showed significant Anti Ulcer property. All the patients were treated regularly with trial medicines during this study there were no complications such as perforation, bleeding etc., were observed in all the 40 cases.

"தொடர்வாத பந்த மலாது குன்மம் வராது"

குன்மம் நோயில் வாதம் அதிகரித்து காணப்படும். வாதத்தை சமப்படுத்தும் சுவைகள் இனிப்பு, புளிப்பு, உப்பு.

Ì ý Á¡¾ ÝÃ½õ துவர்ப்பு சுவையாக, மண் + வளி பூத கூறுபாடு உடையதாக இருப்பினும் குன்ம நோயை குணப்படுத்துவதால் ஒப்புரையின் அடிப்படையில் செயல்படுகிறது.

ÓÍÓÍì ¨ ¸ இளகம் ***கார்ப்பு*** சுவையாக, வளி + தீ பூதகூறுபாடு உடையதாக இருப்பதால் வாதத்தை குறைத்து குன்ம நோயை குணப்படுத்தும். எனவே, இது ஒப்புரையின் அடிப்படையில் செயல்படுகிறது.

Bio-Chemical & Microbiological analysis

Bio-Chemical analysis shows that the trial medicines Gunmathi Choornam is having acid radicals such as sulphide, Phosphate, Carbonates and oxalate, basic radicals such as ferrous iron, Calcium.

The Bio-Chemical analysis of Musumusukai Lehyam shows that it has acid radicals such as sulphide, Phosphate, Fluoride and oxalate. Basic radicals such as ferrous Iron, and calcium.

The antimicrobial study of Gunmathi Choornam and Musumusukai lehyam shows that is highly sensitive against *Staphylococcus aureus, Bacillus subtilis, Streptococcus faecalis, Pseudomonas aeruginosa, Salmonella typhi, Escherichia coli,* and *Candida albicans.*

Pharmacological reports:

Gunmathi chooranam+ Musumusukai Lehyam in combination exhibited anti ulcer activity in pylonic rats model for screening anti ulcer drugs.

Toxicological evaluation

The trial drug Gunmathi Choornam & Musumusukai lehyam did not exhibit any significant toxicity. The drug Gunmathi Choornam and Musumusukai lehyam falls under class 4. (L.D.50>2000 mg/kg). The animals did not show any signs of acute toxicity and behavioural changes.

Bio-Statistical study

The both subjective and objective parameters were statistically significant.

Out of 40 cases 75% had improved, 25% had symptomps relieved.

The statistical analysis of the results obtained from the clinical study were very much encouraging.

Summary

SUMMARY

The pathogenesis of **ERI Gunnam**, clinical features differential diagnosis, prognosis and routine treatment of **ERI Gunnam** mentioned in different Siddha literature were collected.

The pathogenesis of **ERI Gunmam** resembles the pathogenesis of peptic Ulcer in this study modern investigation were used with a view to understand and explain the disease **ERI Gumnam** with modern interpretations.

The 40 patients were examined clearly by both Siddha and modern aspects.

The more prone age group was between 41 to 50^{th} year i.e. in the Pitha kalam

Among 40 cases the disease was found predominant among the non-vegetarians, Alcoholism and found frequently in people of low income group than rich persons.

Most of all the patients have abdominal discomfort, pain related to food, pain in the abdominal region, Diarrhoea, loss of weight.

Among the 40 cases, Maximum no of cases came from Neithal Nilam and the Paruva kalam does not interfere in these studies.

In this study most of the patients had reported to belongs to 'O' group advised to have regular diet control and physical activities, in no cases, side effects and complications were observed.

The preparation of the medicines are easy and economical. The effect of the medicines is proved powerful in greater appeals.

Conclusion

CONCLUSION

The Well known common Gastro intestinal disorder **ERI GUNNAM** was studied in all aspects. All the cases were treated with trail medicines **GUNMATHI CHOORNAM** and **MUSUMUSUKAI LEHYAM** which was found to be free from side effects.

In this study the results and observation showed that 75% cases improved, 25% of the cases symptoms relived.

- Pharmacological study was done for its anti ulcer activity. The results showed GUNMATHI CHOORNAM + MUSUMUSUKAI LEHYAM in combination exhibited activity.
- The drug combination in therapeutic doses did not show evidence of toxicity in haematology and biochemical marker levels or liver and kidney functions.
- The antimicrobial study proved it to be effective against various bacteria.
- Phytochemical tests were done which showed the presence of sulphate, Phosphate, Carbonates, Oxalate, Ferrous, Iron, Calcium.Alkaloids, Flavonoids, Saponins, Glycosides, Carbohydrates, Aminoacids, Triterpenoids.
- The preparations of the medicines are easy and economical. The effect of the medicines is proved powerful in superior appeals.
- The drug is easily edible and the patients did not complaint any difficulty in taking the drug.

ANNEXURE-I

Preparation and Properties of Trial Medicines

PREPARATIONS OF THE TRIAL MEDICINES

DRUG NAME : GUNMATHI CHOORANAM

LITERATURE REFERENCE: « ¸ò¾Â÷ « ð¼Å ¨ ½ Å¡ ¸¼õ À ì ¸õ-98 (Ì ý Á¡¾ Ý Ã ½ õ)

INCREDIENTS OF GUNMATHI CHOORANAM

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Í ì Ì - 10.5 ¸ 3 ÀÄõ

¦ ÀÕ í ¸¡Âõ 4.0 ¸

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தீரும்நோய்கள்: குன்மம், º ò¾ குன்மம், ÅÂüÚ þº× தீரும்.

PROPERTIES OF INREDIENTS IN TRIAL DRUGS:

1. ***கொடிவே-*** : **PLUMBAGO ZEYLANCIA**

Family : Plambaginacia

சுவை : ***கார்ப்பு.***

தன்மை : ***வெப்பம்***

பிரிவு : ***கார்ப்பு***

<u>பொது குணம்</u>

ஒடுமே விப்புருதி ஒடுகின்ற குன்மமெட்டும்

நாடுதனில் மேகரணம் நாடாது - மாடுகளே

காடுதனில் மூ- கையை காணாமலேனலைவீர்

வீட்டி- ருந் திம்முறைசெய் மின்

-குணபாடம் மூ- கை வகுப்பு பக்கம் எண்,271

பொருள் : எண் வகை குண்மம் தீரும்

2. ***சிவதை வேர்*** : **(IPOMEA TURPETHUM)**

Family : Convulaceae

சுவை : ***கைப்பு.***

தன்மை : ***வெப்பம்***

பிரிவு : ***கார்ப்பு***

<u>பொது குணம்</u>

காரமுடன் கைப்பும் உண்டாங் காண எ- விடத்தை

பாரஅனற் குன்மத்தை பற்றறுக்கும் - பாரில்

தெரிஞ்சுரைத்தோம் பேதிதருந் தேமொழியே

கருஞ்சிவதை கந்தம்அது காண்

-குணபாடம் மூ- கை வகுப்பு பக்கம் எண், 99

பொருள் : சிவதை வேரை சுத்திகரித்து பொடித்து தர எரிகுன்மத்தைப் போக்கும்,

3. ***கர்கடக சிங்கி*** : **(RHUS SUCCEDENA)**

Family : Anacardiacea

சுவை : ***துவர்ப்பு***

தன்மை : ***வெப்பம்***

பிரிவு : ***கார்ப்பு***

<u>பொது குணம்</u>

மூலவிரத்தங் கடுப்பு மோதிரத்த பித்தங்கால்

மேல் அடரும்மேகவிரண வெப்போ - டாலத்

துடுங்காங் கொதிப்பு மறுந் தோகை மட மாதே

கடுக்காயின் பூவை கருது

-குணபாடம் மூ- கை வகுப்பு பக்கம் எண், 165

பொருள் : மிகுந்த துவர்ப்பை உடையதால் உள் இரணங்களை ஆற்றும்,

4. *திப்பி-* : **PIPER LONGUM**

Family : Piperaceae

சுவை : *இனிப்பு*

தன்மை : *சீதம்*

பிரிவு : *இனிப்பு*

செய்கை : *அகட்டுவாயகற்றி*

பொது குணம்

இருமல் குன்மம் இரைப்பு கயப்பிணி
ஈளைபாண்டு சந்நியாசம் அரோசகம்
பொருமல் ஊதை சிரப்பிணி மூர்ச்சை நோய்
பூரிக் குஞ்சல தோடம் பி- சமும்
வருமல பெருக்கோடு மகோதரம்
வாதம் ஆதிமுத் தோடன் சுரங்குளிர்
பெருமலைப்புரி மேக பிடகமும்
பேருந் திப்பி- பேரங்கு குரைக்கவே

-குணபாடம் மூ- கை வகுப்பு பக்கம் எண், 368

பொருள் : குன்மம். பாண்டு. அரோசகம். கிருமிநோய் போகும்

5. *ஓமம்* : **CARUM CAPTICUM**

Family : Umbelliferac

சுவை : *கார்ப்பு*

தன்மை : *வெப்பம்*

பிரிவு : *கார்ப்பு*

செய்கை : *அகட்டுவாயகற்றி. அழுகலகற்றி. துவர்ப்பி. பசித்தீ தூண்டி இசிவகற்றி.* (antispasmatic)

பொது குணம்

சீதசுரங் காசஞ் செரியா மந்தம் பொருமல்
பேதியிரைச்சல் கடுப்பு பேராமம் - ஒதிருமல்
பல்லொரு பலமூலம் பகமிவை நோயென் செயுமோர்
சொல்லொடுபோம் ஓமமென சொல்

குணபாடம் மூ- கை வகுப்பு பக்கம் எண், 119

பொருள் : செரியா மந்தம். வயிற்றுப் பொருமல். பேதி. குட- ரைச்சல். வாயிற்கடி பற்றிய வ- போகும்,

6. **சுக்கு** : **ZINGIBER OFFICINALIS**

Family : Zingiberaceae

சுவை : கைப்பு.

தன்மை : வெப்பம்

பிரிவு : இனிப்பு

செய்கை : பசித்தூண்டி. அகட்டுவாயகற்றி

பொது குணம்

சூலை மந்தம் நெஞ்செரிப்பு தோடம் ஏப்பம் அழலை

மூலம் இரைப்பிருமல் மூக்குநீர் - வாலகப

தோடமதி சாரந் தொடர்வாத குன்மம் நீர்த்

தோடம் ஆமம் போக்குஞ் சுக்கு

குணபாடம் மூ- கை வகுப்பு பக்கம் எண், 337

பொருள் : வயிற்றுவ- . நெஞ்செரிவு. ஏப்பம். குன்மம். வயிற்றைப் பற்றிய நோய்கள் தீரம்,

7. **பெருங்காயம்** : **FERULA ASAFOETIDA**

Family : Apiclceac

சுவை : கைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

பொது குணம்

தந்தவேததந்த மூலத்தெழும் பிணி

சருவகாளம் விருச்சிகங் கீடம்மா

மந்தவாதம் உதாவர்த்தம் அல்குல்நோய்

மார்பணங்கட்ட குன்மம் மகோதரம்

உந்து கெர்ப்பத்தின் வித்திரச் சூலைச்சூர்

உதிரபூச்சி சிலேத்துமத்தறும் வ-

வந்தமெய் கடுப் போடிவை முற்றுமே

மாயுநாலுநற் காயங் கிடைக்கினே

குணபாடம் மூ- கை வகுப்பு பக்கம் எண், 508

பொருள் : எண்வகை குன்மம் சூலை. கிருமி. அசீரணம் போகும்,

PREPARATIONS OF THE TRIAL MEDICINES

DRUG NAME : MUMUSUKAI LEHYAM

LITERATURE REFERENCE:

« Û§À¡¸ ¨ Åò¾¢Â ¿Å½¾õ Àì¸õ - 82. (1139.Ó Í Ó Í ì ¨ ¸ þÇ¸õ)

INCREDIENTS OF MUMUSUKAI LEHYAM

Ó Í Ó Í ì ¨ ¸ º¡Ú	-	1/2 ÀÊ
¦ÅøÄõ	-	3 ÀÄõ. (105 ¸¢Ã¡õ)
§Áüò§¾¡ø º¦¾¢Â Í ì Ì	-	1 ÀÄõ. (35 ¸¢Ã¡õ)
Á¢Ç Ì	-	1 ÀÄõ. (35 ¸¢Ã¡õ)
« ¡¢º¢ò ¾¢ôÀ¢Ä¢	-	1 ÀÄõ. (35 ¸¢Ã¡õ)
² Ä¡¢º¢	-	1 ÀÄõ. (35 ¸¢Ã¡õ)
¿üº¦Ã¸õ	-	1 ÀÄõ. (35 ¸¢Ã¡õ)

புறெப்ழி

சுக்கை மேல் தோல் சீவிக்கொண்டு பின் அதனுடன் மிளகு. திப்பி- . ஏலரிசி. சீரகம் ஆகியவற்றை இடித்து துணியில் ச- த்துக் கொண்டேன், முசுமுசுக்கைச் சாற்றில் வெல்லத்தைக் கரைத்து அடுப்பேற்றி பாகு பதத்தில் மேற்படி தூளை கொட்டி கிளறி இறக்கி ஆறின பின் பீங்கான் பாத்திரத்தில் வைத்து எட்டு நாள் தானிய புடம் வைத்து எடுத்து பத்திரப் படுத்திக் கொண்டேன்,

அளவு - 5 கிராம். இருவேளை

தீரும் நோய் : எரிகுன்மம்

1. ***முசுமுசுக்கை*** : **BROYONIA SCABRA**

   Family : Cucurbitaceae

   சுவை : ***துவர்ப்பு***

   தன்மை : ***வெப்பம்***

   பிரிவு : ***கார்ப்பு***

<u>பொது குணம்</u>

கந்தம் பரவுகளி சளியும் புஜ விடமும்

மந்தம் பெறுவிடுமும் வாந்திகளும் - அந்தம

பெருகுருங்குக் கிச்சூடும் பித்தமுமிருங்கா

§ÚÏWe¡ t úLRôu

குணபாடம் மூ- கை வகுப்பு பக்கம் எண், 561

2 . சர்க்கரை : **SACCHARUM OFFICINARUM**

Family : Poaceace

சுவை : இனிப்பு,

தன்மை : சீதம்

பிரிவு : இனிப்பு

செய்கை : அழுகலகற்றி, உள்ளழலாற்றி, உடல் உரமாக்கி

பொது குணம்

குன்ம பித்தம் போக்குமதி கோழைதனை யுண்டாக்கும்

துன்மலத்துட் கீடத்தை தோற்றுவிக்கும் – நன்மை போல்

மெல்லமது நீரை விளைவிக்கும் மாமதுர

வெல்லமென நாளும் விளம்பு

குணபாடம் மூ– கை வகுப்பு பக்கம் எண். 166

பொருள் : குன்மத்தைப் போக்கும்

3. சுக்கு : **ZINGIBER OFFICINALIS**

Family : Zingiberaceae

சுவை : கைப்பு,

தன்மை : வெப்பம்

பிரிவு : இனிப்பு

செய்கை : பசித்தூண்டி, அகட்டுவாயகற்றி

<u>பொது குணம்</u>

சூலை மந்தம் நெஞ்செரிப்பு தோடம் ஏப்பம் அழலை

மூலம் இரைப்பிருமல் மூக்குநீர் – வாலகப

தோடமதி சாரந் தொடர்வாத குன்மம் நீர்த்

தோடம் ஆமம் போக்குஞ் சுக்கு

குணபாடம் மூ– கை வகுப்பு பக்கம் எண்.

பொருள் : வயிற்றுவ– , நெஞ்செரிவு, ஏப்பம், குன்மம், வயிற்றைப் பற்றிய நோய்கள் தீரும்.

4 . மிளகு : **PIPER NIGRUM**

Family : piperaceac

சுவை : கைப்பு, கார்ப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

செய்கை : அகட்டுவாயகற்றி (Carminative)

<u>பொது குணம்</u>

சீதசுரம் பாண்டு சிலேத்மங் கிராணி குன்மம்
வாதம் அருசி பித்தம் மாமூலம் – ஒது சன்னி
அபஸ்மாரம் அடன்மேகம் காசமிவை
நாசங் கறிமிளகினால்

குணபாடம் மூ– கை வகுப்பு பக்கம் எண். 761

பொருள் : குன்மம், வாயு, சுவையின்மை, செரியாமை இவை போகும்.

5. திப்பி– : **PIPER LONGUM**

Family : Piperaceae

சுவை : இனிப்பு

தன்மை : சீதம்

பிரிவு : இனிப்பு

செய்கை : அகட்டுவாயகற்றி

பொது குணம்

இருமல் குன்மம் இரைப்பு கயப்பிணி
ஈளைபாண்டு சந்நியாசம் அரோசகம்
பொருமல் ஊதை சிரப்பிணி மூர்ச்சை நோய்
பூரிக் குஞ்சல தோடம் பி– சமும்
வருமல பெருக்கோடு மகோதரம்
வாதம் ஆதிமுத் தோடன் சுரங்குளிர்
பெருமலைப்புரி மேக பிடகமும்
பேருந் திப்பி– பேரங்கு குரைக்கவே

குணபாடம் மூ– கை வகுப்பு பக்கம் எண். 368

பொருள் : குன்மம், பாண்டு, அரோசகம், கிருமிநோய் போகும்

6 . *ஏ ல ரி சி* : **ELETARIA EARDAMUM**

Family : Zingi Beraceace

சுவை : கார்ப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

செய்கை : அகட்டுவாயகற்றி, வெப்பமுண்டாக்கி, பசித்தூண்டி

பொது குணம்

தொண்டை வாய்கவுள் தாலு குதுங்களில்
தோன்றும் நோய் அதிசாரம் பன்மேகத்தால்
உண்டைபோல் எழுங் கட்டி கிரிச்சரம்
பண்டை வெக்கை வித்தாக நோய் காசமும்

பாழுஞ்சோமம் பிணி விந்து நட்டம் உள்

அட்டையீளை வன்பித்தம் இவைக்கெல்லாம்

ஆலமாங்கமழ் ஏலமருந்தே

குணமாடம் மூ– கை வகுப்பு

குணபாடம் மூ– கை வகுப்பு பக்கம் எண். 114

பொருள் : வாந்தி, பித்தநோய், வயிற்று பிணிகள் தீரும்.

7. **சீரகம்** : **Cuminum cyminum**

Family : Umbliferaceae

சுவை : கார்ப்பு, இனிப்பு

தன்மை : தட்பம், பிரிவு : இனிப்பு

செய்கை : அகட்டு வாய் வகற்றி

பசித்தீத்தூண்டி

துவர்ப்பி

வெப்பமுண்டாக்கி

குணம்:

பித்தமெனு மந்திரியைப் பின்னப் படுத்தியவன்

சத்துருவை யுற்துறந்து சாதித்து மத்தனெனும்

ராசனையு மீவென்று நண்பைப் பலப்படுத்தி

போசனகு டாரிசெயும் பேரா (தேரன் வெண்பா)

‘‘வாந்தி யருசிகுன்மம் வாய்நோய்பீ லிகமிரைப்

பேற்றிருமல் கல்லடைப்பி லாஞ்சனமுட் – சேர்ந்தகம்மல்

ஆசனடு பாரியெனும் அந்தக் கிரகணியும்

போசனகு பாரியுண்ணப் போம்.

‘‘சீரகத்தைப் பொடித்து வெண்ணெயில்

கொடுக்க எரிகுன்மம் போகும்’’.

குணபாடம் – மூலிகை வகுப்பு ப.எண். 460

பொருள் : தீக்குற்றத்தை தன்னிலைப்படுத்தி, வயிற்றின் மந்தத்தைப் போக்கி, பசியை உண்டாக்கி, உணவைச் செரிக்குமாறு செய்யும்.


MUSUMUSUKAI


SIVADHAI


KARKADAGASINGI


KODIVELI ROOT


THIPPILI


CHUKKU


SEERAGAM


PERUNGAYAM


MILAGU


ELLARASI


VELLAM


OMAM

ANNEXURE-II

Anti Microbial Activity

Introduction

The development of bacterial resistance to presently available antibiotics has necessitated the search for new antibacterial agents[11]. The Gram-positive bacteria such as *Staphylococcus aureus* are mainly responsible for postoperative wound infection, toxic shock syndrome and food poisoning. The Gram-negative bacterium such as *E. coli* is present in human intestine and causes lower urinary tract infection, coleocystis or septicemia. The Gram positive and Gram-negative bacteria can be inhibited by antibiotics, either by blocking the protein synthesis or peptidoglycan synthesis in bacterial cell wall. The indiscriminate use of chemical pesticides has given rise to serious environmental pollution, genetic resistance of pests, toxic residues in stored products and hazards from handling etc. Therefore, there is a need to develop safe pesticides which are effective, biodegradable, broad-spectrum of activity and do not leave any harmful effect on environment. In order to identify such a drug an attempt has been made with Musumusukai Legium and Gunmathi chooranam-a siddha drug.

Drug Material:

Musumusukai Legium and Gunmathi chooranam were collected from Chennai. Tamil nadu, India. Quality was identified and analysed in the department of medicine, Anna hospital, Govt siddha medical college, Arumbakkam, Chennai.

Preparation Of stock solution:

For the preparation of various concentrations of stock solutions of the siddha drugs Musumusukai Legium and Gunmathi chooranam were used in the present study. it was dissolved separately with 50 ml of DMSO under reduced pressure. The condensed supernatant was kept at 4 ºC prior to test.

Test microorganisms:

Strains, including fungi and bacteria were obtained from Persian Type Culture Collection (PTCC). The microorganisms used in the present study include *Staphylococcus aureus, Bacillus subtilis, Streptococcus faecalis, Aspergillus niger, Escherichia coli,* and *Candida albicans*.

Experimental

Suitable strains of above-mentioned microorganisms were procured from the microbiology laboratory of our institute. Antibacterial and antifungal activities were studied by agar well plate method. The zone of inhibition of the Musumusukai Legium and Gunmathi chooranam was performed at concentrations of 25, 50 and 100 mcg/ml of the fractions in dimethyl sulphoxide (DMSO). Ciprofloxacin (5 mg/ml) and Clotrimazole (25 mg/ml) were used as reference controls for the antibacterial and antifungal studies, respectively. Solvent control (only DMSO) was also maintained throughout the experiment.

Results And Discussion

The Musumusukai Legium was found to be more potent than the Gunmathi chooranam. Zone of inhibition study reveals that both Musumusukai Legium and Gunmathi chooranam showed antibacterial and antifungal activity in a dose dependant manner and was comparable with the standard drugs. The results also reveal that both the drugs were more active against *Streptococcus faecalis* and less active against *Basillus subtilis*.

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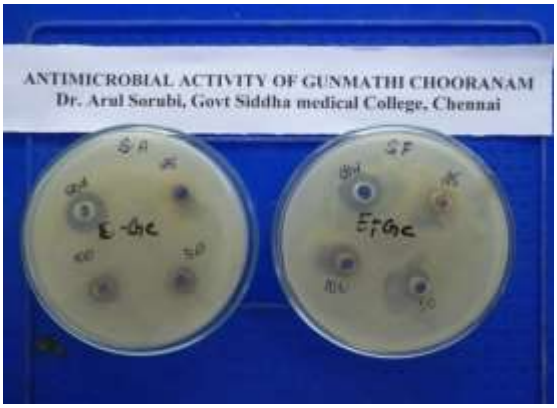
ANTIMICROBIAL ACTIVITY OF GUNMATHI CHOORANAM
Dr. Arul Sorubi, Govt Siddha medical College, Chennai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ANTIMICROBIAL ACTIVITY OF MUSUMUSUKAI LEGIUM
Dr. Arul Sorubi, Govt Siddha medical College, Chennai


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ANNEXURE-III

Phyto Chemical Analysis

PHYTO - CHEMICAL ANALYSIS

CHEMICAL ANALYSIS OF TRIAL MEDICINES

Preperation of Sodium Carbonate extract

2gm of Trial medicines **Gunmathi choornam** – drug – I and **Musumusukai Lehyam**-drug-II are weighed accurately and mixed with 5gm of sodium carbonate taken in a 100ml beaker and 20ml of distilled water is added. The solution is boiled for 10 minutes, coold and then filtered. The filterate is called sodium Carbonate extract.

S. NO	EXPERIMENT	OBSERVATION		INFERENCE	
		Drug I	**Drug II**	**Drug I**	**Drug II**
1.	**TEST FOR ACID RADICALS**				
a)	**Test for Sulphate** 2 ml of the above prepared extract is taken in a test tube. To this add 2 ml of 4% Ammonium oxalate solution.	White precipitate is obtained	White precipitate	Presence of sulphate	Presence of sulphate
b)	2 ml of extract is added with 2 ml of dilute Hydrocholoric acid until the effervescence ceases off. Then 2 ml of Barium chloride solution is added.	Present	Present	Present	Present
2.	**Test for Chloride** 2 ml of extract is added with dilute Nitric acid till the effervescence ceases. Then 2 ml of silver Nitrate solution is added.	No white precipitate	No white precipitate	Absence of Chloride	Absence of Chloride
3.	**Test for Phosphate** 2 ml of the extract is treated with 2 ml of Ammonium Molybdate solution and 2 ml of concentrated Nitric Acid.	yellow precipitate is formed	yellow precipitate is formed	Presence of phosphate	Presence of phosphate
4.	**Test for Carbonate** 2 ml of the extract is treated with 2 ml of Magnisium Sulphate solution.	Effervesce nce formed	Effervesce nce is formed	Presence of Carbonate	Presence of Carbonate
5.	**Test for Sulphide** 1 gm of the substance is treated with 2 ml of concentrated Hydrocholric acid	Absent	Absent	Absent	Absent
6.	**Test for Nitrate** I gm of the substance is heated with copper turnings and concentrated Suophuric acid and viewed the test tube vertically down.	Absent	Absent	Absent	Absent

7a.	**Test for Fluoride and Oxalate** 2 ml of the extract is added with 2 ml of dilute Acetic acid and 2 ml of Calcium Chloride solution and heated.	Absent	Present	Absent	Present
b.	5 drops of clear solution is added with 2 ml of dilute sulphuric acid and slightly warmed. To this, 1 ml of dilute Potassium Permanganate solution is added.	Present	Present	Present	Present
8.	**Test for Nitrite** 3 drops of the extract is placed on a filter paper. On that, 2 drops a Acetic Acid and 2 drops of Benzidine solution is placed.	Absent	Absent	Absent	Absent
9.	**Test for Borate** 2 pinches of the substance is made into paste by using Sulphuric acid and Alcohold (95%) and introduced into the blue flame.	Absent	Absent	Absent	Absent
II.	**TEST FOR BASIC RADICALS**				
10.	**Test for lead** 2 ml of the extract is added with 2 ml of Pottassium lodide solution	Absent	Absent	Absent	Absent
11a	**Test for Copper** One pinch of substance is made into paste with concentrated Hydrochloric acid in a watch glass and introduced into the non luminous part of the flame.	Absent	Absent	Absent	Absent
b.	2ml of the extract is added with excess of Ammonia solution	Absent	Absent	Absent	Absent
12.	**Test for Aluminium** To the 2nd ml of extract. Sodium Hydroxide solution is added in drops to excess.	Absent	Absent	Absent	Absent

13a	**Test for Iron** To the 2nd ml of extract, 2 ml of Ammonium Thiocyanate solution is added.	Present	Present	Present	Present
b.	To the 2nd ml of extract, 2 ml of Ammonium Thiocyanate solution and 2 ml of concentrated Nitric Acid is added.	Present	Present	Present	Present
14.	**Test for Zinc** To the 2 ml of extract Sodium Hydroxide solution is added in drops to excess.	Absent	Absent	Absent	Absent
15.	**Test for Calcium** 2 ml of the extract is added with 2 ml of 4% Ammonium Oxalate solution.	Present	Present	Present	Present
16.	**Test for Magnesium** To 2ml of extract, Sodium Hydroxide solution is added in drops to excess.	Absent	Absent	Absent	Absent
17.	**Test for Ammonium** To 2 ml of extract few ml of Nessler's Reagent and excess of Sodium Hydroxide solution are added.	Absent	Absent	Absent	Absent
18.	**Test for Potassium** A pinch of substance is treated with 2 ml of Sodium Nitrite solution and then treated with 2 ml of Cobal Nitrate in 30% glacial Acetic acid.	Absent	Absent	Absent	Absent
19.	**Test for Sodium** 2 pinches of the substance is made into paste by using Hydrocholoric acid and introduced into the blue flame.	Absent	Absent	Absent	Absent
20.	**Test for Mercury** 2 ml of the extract is treated with 2 ml of Sodium Hydroxide solution.	Absent	Absent	Absent	Absent
21.	**Test for Arsenic** 2 ml of extract is treated with 2 ml of silver Nitrate solution	Absent	Absent	Absent	Absent

Results

The given sample contains

ACID RADICALS

1. Gunmathi Choornam

    - Sulphate
    - Phosphate
    - Carbonate
    - Oxalates

2. Musumusukai Lehyam

    - Sulphate
    - Phosphate
    - Fluoride
    - oxalate

BASIC RADICALS

1. Gunmathi Choornam

    - Ferrous Iron
    - Calcium

2. Musumusukai Lehyam

    - Ferrous iron
    - Calcium

SOPHISTICATED ANALYTICAL INSTRUMENT FACILITY
INDIAN INSTITUTE OF TECHNOLOGY, MADRAS
Chennai - 600 036. INDIA

CERTIFICATE

Certified that herbal drugs Gunmathi Chooranam and Musumusukai Legiyam formulated by Dr.S.Arul Sorubi, III Year M.D(S) (Maruthuvam), Government Siddha Medical College, Chennai-106, are analysed by Physico-Chemical, SEM,ICP and Phyto Chemical Methods at SAIF, IITM, Chennai-36, during August 2011.

Dr. R. MURUGESAN
Scientific Officer Gr.-I
Sophisticated Analytical Instrument Facility
Indian Institute of Technology, Madras
Chennai-600 036

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e-mail : saif@iitm.ac.in http://www.saif.iitm.ac.in

Table-1.

Colour characters of Gunmathi Chooranam.

S No	Solvent used	Under ordinary light	Under ultra violet light
1	PPM	Light brown	Light brown

PPM-Powdered plant material

Table-2.

Physicochemical properties of Gunmathi Chooranam.

S No.	Parameters	Values obtained (%w/w)	Heavy/ toxic metals	
1	Total ash value	9.21	Lead	BDL
2	Acid insoluble ash	20.95	Cadmium	BDL
3	Water soluble ash	5.60	Mercury	BDL
4	Moisture content	9.2	Arsenic	BDL
5	Foreign organic matter	8.4	Volatile oil	BDL
6	Crude fibre content	16.00		
7	Alcohol soluble extractive	3.5		
8	Water soluble extractive	13.6		

Table-3.

Colour, nature and percent yields of extracts of Gunmathi Chooranam.

S.no.	Extract Solvents	Colour	TLC/GC (PEAKS)	Nature	% Yield(w/w)	SEM-Micro graph partical size range in micron	pH
1	Water	Light brown	6	Solid	37	1-3 micron	7.3-7.6

Table-4.

Preliminary phytochemical Studies on extracts of Gunmathi Chooranam.

S.no	Phytoconstituents	Aqueous
1	Alkaloids	+
2	Flavonoids	+
3	Saponins	+
4	Glycosides	+
5	Carbohydrates	+
6	Triterpenoids	+

+ = Present, – = Absent.

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PERKIN ELMER OPTIMA 5300DV ICP-OES

SampleID	Analyte	Mean
GC		
	As193.696	BDL
	Cd 226.502	BDL
	Hg253.652	BDL
	Ca 317.933	20.12mg/L
	Fe 238.204	4.670 mg/L
	K 766.490	210.21 mg/L
	Na 589.592	27.200 mg/L
	P 213.617	35.60mg/L
	Pb 230.204	BDL
	S 181.975	14.20mg/L

BDL=Below detection limit

Table-1.

Colour characters of Musumusukai Legiyam.

S No	Solvent used	Under ordinary light	Under ultra violet light
1	PPM	Dark brown	Dark brown

PPM-Powdered plant material

Table-2.

Physicochemical properties of Musumusukai Legiyam.

S No.	Parameters	Values obtained (%w/w)	Heavy/ toxic metals	
1	Total ash value	8.37	Lead	BDL
2	Acid insoluble ash	0.95	Cadmium	BDL
3	Water soluble ash	7.2	Mercury	BDL
4	Moisture content	8.78	Arsenic	BDL
5	Foreign organic matter	8.4	Volatile oil	BDL
6	Crude fibre content	35.00		
7	Alcohol soluble extractive	6.2		
8	Water soluble extractive	11.25		

Table-3.

Colour, nature and percent yields of extracts of Musumusukai Legiyam.

S.no.	Extract Solvents	Colour	TLC/GC (PEAKS)	Nature	% Yield(w/w)	SEM-Micro graph partical size range in micron	pH
1	Water	Dark brown	7	Solid	42	2-1.2 micron	7.1-7.3

Table-4.

Preliminary phytochemical Studies on extracts of Musumusukai Legiyam.

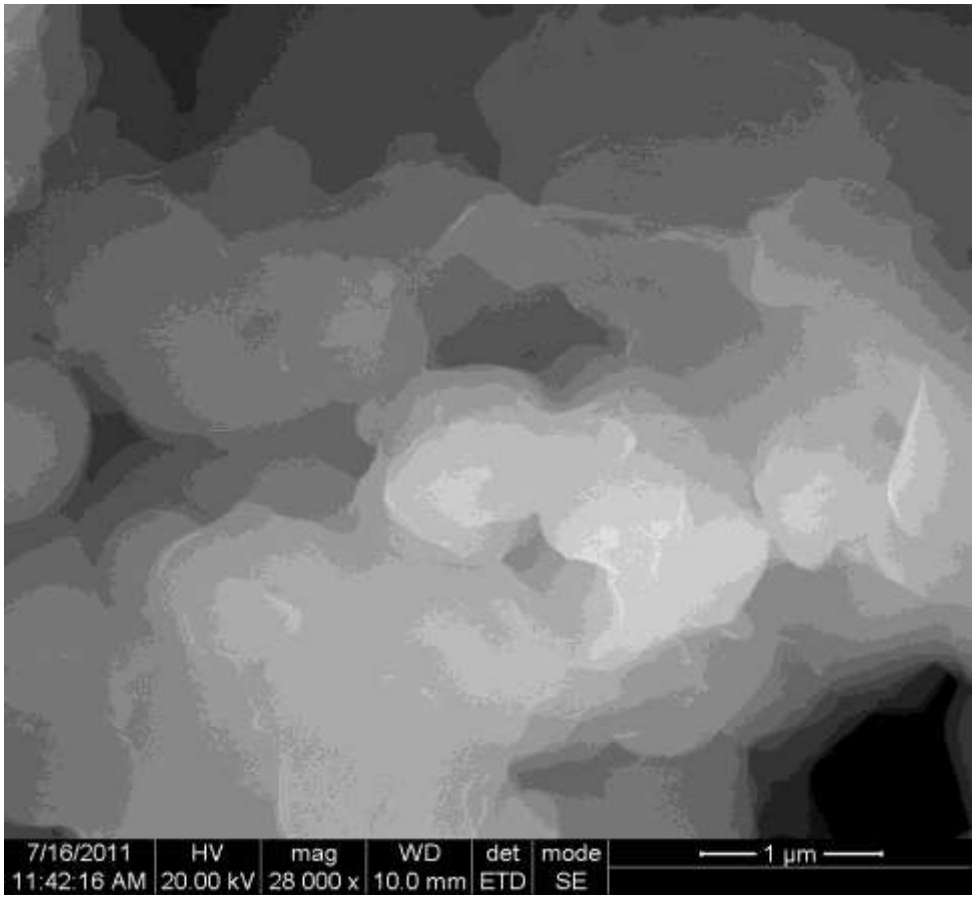
S.no	Phytoconstituents	Aqueous
1	Alkaloids	+
2	Flavonoids	+
3	Saponins	+
4	Glycosides	+
5	Carbohydrates	+
6	Amino acids	+
7	Triterpenoids	+

+ = Present, − = Absent.

SOPHISTICATED ANALYTICAL INSTRUMENT FACILITY
IITM,CHENNAI-36
PERKIN ELMER OPTIMA 5300DV ICP-OES

SampleID	Analyte	Mean
ML		
	As193.696	BDL
	Cd 226.502	BDL
	Hg253.652	BDL
	Ca 317.933	18.41mg/L
	Fe 238.204	3.712mg/L
	K 766.490	180.251 mg/L
	Na 589.592	13.150 mg/L
	P 213.617	31.250mg/L
	Pb 230.204	BDL
	S 181.975	17.14mg/L

BDL=Below detection limit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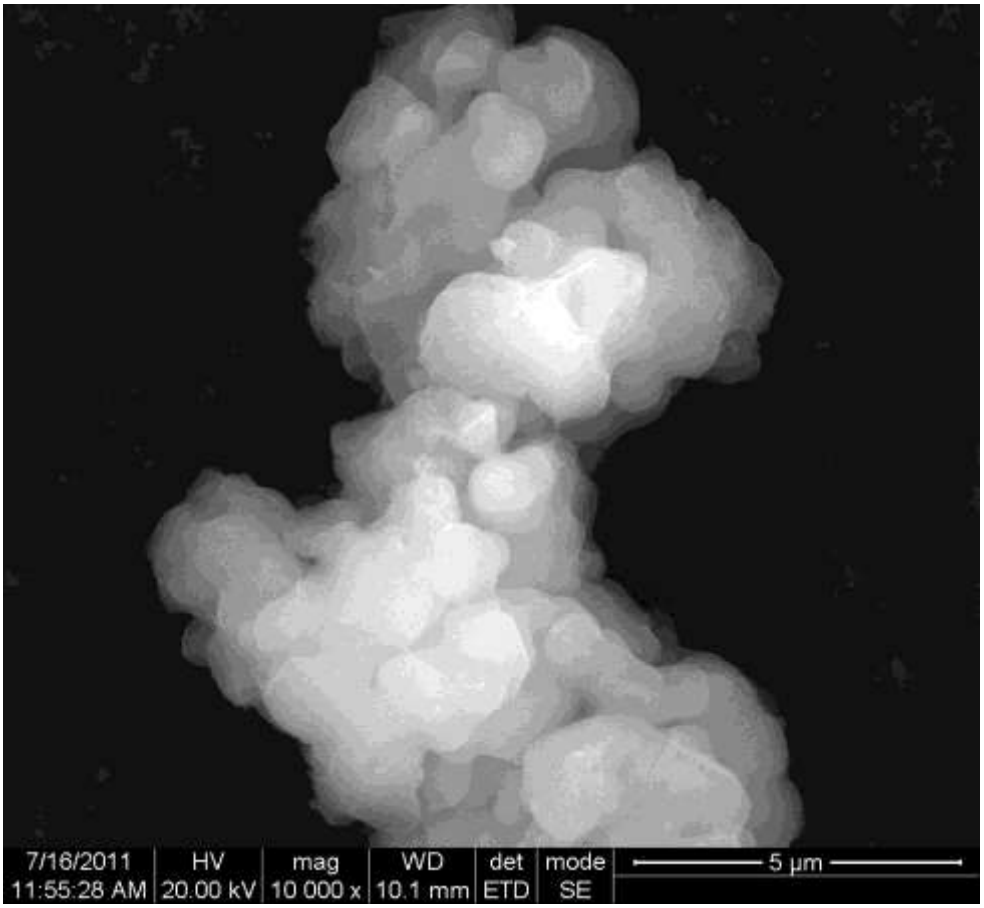
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ANNEXURE-IV

Statistical Analysis

I Treatment for ERI Gunmam (Peptic Ulcer)

The most popular statistical tool, namely, chi-square (Good ness of fit) analysis has been employed to analyses the effectiveness with the help of a Hypothesis.

Hypothesis:

There is significant improvement among the patients for the treatment of ERI Gunmam.

Table of observed frequencies 'O'

Symptoms	**No of patients**		**Total**
	Improved 'O'	**Not improved**	
Epigastric Pain	19	1	20
Heart burn	8	4	12
Abdominal discomfort	26	4	30
Diarrhea	8	0	8
Loss of weight	3	1	4
Total	**64**	**10**	**74**

1Table of Expected frequencies "E"

Symptoms	**'O'**	**'E'**
Epigastric Pain	19	17.29
Heart burn	8	10.38
Abdominal discomfort	26	25.95
Diarrhea	8	6.92
Loss of weight	3	3.46
Total	**64**	**64.00**

Calculation of X^2

O	E	(O-E)	$(O-E)^2$	$(O-E)^2 / E$
19	17.29	1.703	2.900	0.617
8	10.38	-2.378	5.654	0.544
26	25.95	0.055	0.003	1.16593E-4
8	6.92	1.082	1.700	0.169
3	3.46	-0.459	0.210	0.060
			Total	**0.94**

Result:

The calculated value of X^2 is = 0.94.

The table value of X^2 < 5% significant level.

As per probability level (alpha) for 4 df = 9.488.

Inference:

Since the calculated value is less than the table value, the Hypothesis is accepted. Hence it is concluded that the treatment is **effective and significant**.

ANNEXURE-V

Preclinical Pharmacological & Toxicological Study

CERTIFICATE

This is to certify that the project title **"Pharmacological activities of Gunmathi churnam and Musumusukai legium"** has been approved by the IAEC.

XII / VELS / PCOL / 53 / 2000 / CPCSEA / IAEC / 11.03.11

XII / VELS / PCOL / 52 / 2000 / CPCSEA / IAEC / 11.03.11

Name of member secretary of IAEC:
Dr. J. Anbu

Name of CPCSEA nominee:
Dr. K. Sadhasivan pillai

Signature with date

8/7/11

Member Secretary of IAEC:

CPCSEA nominee:

DR.J.ANBU, M.Pharm.,Ph.D.,D.M.L.T., MBA
Professor & Head
Department of Pharmacology & Toxicology
School of Pharmaceutical Sciences
Vels University
Pallavaram, Chennai-600 117

VEL'S COLLEGE OF PHARMACY
Pallavaram, Chennai-117.

VELS INSTITUTE OF SCIENCE, TECHNOLOGY AND ADVANCED STUDIES (VISTAS)

Estd. u/s 3 of the UGC Act, 1956

ATTENDANCE CERTIFICATE

This is to certify that Mr/Ms. Dr S Arul Soruba Post graduate student of Government Siddha Medical College, Arumbakkam, Chennai has carried out his/her project work entitled as "Biological & pharmacological activities of Guvvamathi choornam" in the Department Of Pharmacology, School Of Pharmaceutical Sciences, Vels University, Chennai under my direct supervision and guidance from _____ to _____.

VEL'S COLLEGE OF PHARMACY * Pallavaram, Chennai 117. *

Project Co-Ordinator

DR.J.ANBU, M.Pharm.,Ph.D.,D.M.L.T., MBA
Professor & Head
Department of Pharmacology & Toxicology
School of Pharmaceutical Sciences
Vels University
Pallavaram, Chennai-600 117.

VELS UNIVERSITY

CHENNAI - INDIA

VELS INSTITUTE OF SCIENCE, TECHNOLOGY AND ADVANCED STUDIES (VISTAS)

Estd. u/s 3 of the UGC Act 1956

ATTENDANCE CERTIFICATE

This is to certify that Mr/Ms. Dr. S. Arul Sorubi Post graduate student of Government Siddha Medical College, Arumbakkam, Chennai has carried out his/her project work entitled as "Biological & pharmacological activities of Muthuvaguppukari Legium." in the Department Of Pharmacology, School Of Pharmaceutical Sciences, Vels University, Chennai under my direct supervision and guidance from _____ to _____.

VEL'S COLLEGE OF PHARMACY * Pallavaram, Chennai-117. *

Project Co-Ordinator

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Professor & Head
Department of Pharmacology & Toxicology
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Pallavaram, Chennai-600 117.

MATERIALS AND METHODS

Test Drugs

The following medicinal plants used in the study were collected and processed by the methods prescribed in standard text books of siddha medicines.

Gunmathi chooranam and Musumusukai lehyam

Were prepared by the method described in **(Agathiyar Attavanai Vagadam (Page No: 98)** and **Anuboga vaidtheya navaneetham (Page No: 82))**

Gunmathi chooranam

Drug and Preparation of Stock solution

The aqueous suspension of Gunmathi Chooranam was prepared in 0.5 % carboxymethylcellulose (CMC) solution in distilled water prior to oral administration to animals. It was used within seven days and stored at 8^{o}C while for further use, freshly prepared solution was used. The vehicle alone served as control. All the drugs and chemicals were of analytical grade. Ranitidine (Osaka), Ethanol (Research lab) were used.

Experimental animals

Albino rats (Wistar strain) of either sex, weighing 180-200g and Swiss albino mice (20-22g) of either sex were procured from animal housing facility, School of pharmaceutical Sciences, Vels university, Chennai. All the animals were placed in polypropylene cages in a controlled room temperature 22±1°C and relative humidity of 60-70% in animal house. The animals were maintained on standard pellet diet (Sai meera foods Pvt limited, Bangalore, India) and water *ad libitum*. They were acclimatized to laboratory condition for seven days before commencement of the experiment. Ethical clearance was obtained from Institutional Animal Ethical Committee.

Acute oral toxicity study

Acute oral toxicity study of Gunmathi Chooranam was carried out in Swiss albino mice of both sexes (20-22 g) according to OECD guidelines no 423. Gunmathi Chooranam at different doses up to 2000mg/kg, p.o. was administered and animals were observed for behavioral changes, any toxicity and mortality up to 48 h.

ASSESSMENT OF ANTI-ULCER ACTIVITY

Pyloric ligation induced gastric ulceration.

Albino rats of either sex were divided into five groups of six animals each. Animals were fasted for 24 h before the study, but had free access to water.

Group I: treated with vehicle alone (2ml/kg, p.o.) and was kept as control.

Group II: treated with Ranitidine (100mg/kg, p.o.) and was kept as standard.

Group III: treated with the Gunmathi Chooranam (125mg/kg, p.o.).

Group IV: treated with the Gunmathi Chooranam (250mg/kg, p.o.).

Group V: treated with the Gunmathi Chooranam (500mg/kg, p.o.).

After 1h of drug treatment, they were anaesthetized with the help of anesthetic ether; the abdomen was opened by a small midline incision below the xiphoid process. Pyloric portion of the stomach was slightly lifted out and ligated according to method of Shay et al., avoiding traction to the pylorus or damage to its blood supply. The stomach was replaced carefully and the abdominal wall was closed by interrupted sutures. Rats were sacrificed by an over dose of anaesthetic ether after four hours of pyloric ligation. The abdomen was opened, cardiac end of the stomach was dissected out and the contents were drained into a glass tube. The volume of the gastric juice was measured and centrifuged at 2000 rpm for 10 min. From the supernatant, aliquots (1 ml of each) were taken for the determination of pH, total and free acidity. The inner surface of free stomach was examined for gastric lesions.

Determination of PH

An aliquot of 1ml gastric juice was diluted with 1ml of distilled water and pH of the solution was measured using pH meter. Determination of total acidity

An aliquot of 1ml gastric juice diluted with 1ml of distilled water was taken into a 50ml conical flask and two drops of phenolphthalein indicator was added to it and titrated with 0.01N NaOH until a permanent pink colour was observed. The volume of 0.01N NaOH consumed was noted.

The total acidity is expressed as meq./l by the following formula:

$$n \times 0.01 \times 36.45 \times 1000$$

Where n is volume of NaOH consumed, 36.45 is molecular weight of NaOH, 0.01 is normality of NaOH, 1000 is the factor (to be represented in litre)

Determination of free acidity

Instead of phenolphthalein indicator, the Topfer's reagent was used. Aliquot of gastric juice was titrated with 0.01N NaOH until canary yellow colour was observed. The volume of 0.01N NaOH consumed was noted. The free acidity was calculated by the same formula for the determination of total acidity.

Macroscopic evaluation of stomach

The stomach was opened along the greater curvature, rinsed with saline to remove gastric contents and blood clots and examined by a X5 magnifier lens to assess the formation of ulcers. The number of ulcers were counted. Ulcer scoring was undertaken according to Vogel et al.

Ulcer index was measured by using following formula according to vogel et al.

$$UI = UN + US + UP \times 10 - 1$$

UI= Ulcer Index; UN= Average number of ulcers per animal; US =Average number of severity score; UP=percentage of animals with ulcers

Percentage inhibition of ulceration was calculated as below:

$$\% \text{ Inhibition of Ulceration} = \frac{\text{(Ulcer index Control-Ulcer index Test)}}{\text{Ulcer index Control}} \times 100$$

Ulcer score	Descriptive /observation
0	Normal coloured stomach

0.5	Red colouration
1.0	Spot ulcers
1.5	Haemorrhagic streak
2.0	Ulcers
3.0	Perforation

Statistical analysis

The results are expressed as the mean± SEM for each group. Statistical differences were evaluated using a One-way analysis of variance (ANOVA) followed by Dunnet's t-test. Results were considered to be statistically significant at $P<0.05$.

RESULTS

Acute oral toxicity study

Swiss albino mice of both the sexes treated with Gunmathi Chooranam did not show any behavioral changes, toxic reaction or mortality. It was found to be safe at the dose of 2000mg/kg. LD_{50} of the Gunmathi Chooranam was found to be >2000mg/kg.

Pyloric ligation induced gastric ulceration

Effect of Gunmathi Chooranam on pyloric ligation induced ulceration is shown in table-1. The pyloric ligation has caused the accumulation of gastric secretions with pH 3.88±0.17 in a control group. The total acidity and free acidity of the gastric secretions were found to be 172±2.28 and 115.11±0.48meq./l respectively. Pretreatment with the Gunmathi Chooranam, significantly ($P<0.01$) reduced the volume of gastric secretions 3.00±0.03, 3.51±0.03 and 3.33±0.03ml at the doses of 125, 250 and 500mg/kg respectively. pH of the gastric fluid was significantly ($P<0.05$) elevated up to 4.52±0.46 only at higher dose of the test drug.

In addition, total acidity and free acidity were also reduced significantly ($P<0.01$) in a dose dependant manner. Further it is observed that pyloric ligation has caused gastric ulcerations and pretreatment with Gunmathi Chooranam has reduced them significantly ($P<0.01$) in a dose dependent manner. In this model, percentage inhibition of ulceration was found to be 52, 64 and 66

at 125, 250 and 500mg/kg respectively. The gastroprotection offered by the test drug was comparable to that of the standard drug, ranitidine (100mg/kg).

DISCUSSION

Although in most of the cases the etiology of the ulcers is unknown, it is generally accepted that they are a result of an imbalance between aggressive factors and the maintenance of mucosal integrity through endogenous defensive mechanisms. Pylorus ligation induced ulcers are due to auto digestion of the gastric mucosa and breakdown of the gastric mucosal barrier. These factors are associated with the development of upper gastrointestinal damage including lesions, ulcers and life threatening perforation and hemorrhage. Aspirin, phenylbutazone, indomethacin and some non-steroidal anti-inflammatory drugs are also known to cause duodenal and gastric ulceration. Prostaglandin E_2 and I_2 are predominantly synthesized by the gastric mucosa and are known to inhibit the secretion of gastric acid and stimulate the secretion of mucus and bicarbonate.

Hydrophobic surfactant - like phospholipids secretion in the gastric epithelial cells is also stimulated by the prostaglandin. Volume of gastric secretion is an important factor in the production of ulcer due to exposure of unprotected lumen of the stomach to the accumulating acid. The antiulcer property of Gunmathi Chooranam in pylorus ligation model is evident from its significant reduction in free acidity, total acidity, number of ulcers and ulcer index. Gunmathi Chooranam treated animals significantly inhibited the formation of ulcers in the pylorus ligated rats and also decreased both the concentration and increased the pH, it is suggested that Gunmathi Chooranam can suppress gastric damage induced by aggressive factors. It is suggested that, the active compounds would be able to stimulate mucus, bicarbonate and the prostaglandin secretion and counteract with the deteriorating effects of reactive oxidants in gastrointestinal lumen. So the antiulcer activity of Gunmathi Chooranam may be attributed to its active principle.

CONCLUSION

The results obtained in the experimental model of pylorus ligation induced ulceration method in rats. The Gunmathi Chooranam was found to posses remarkable ulcer protection of 68.2% at 500mg/Kg and standard drug at 68.4%. Pylorus ligation consistently caused hemorrhagic lesions in the mucosa of the glandular stomach, indicating true ulcer formation as stated in histological findings. Pretreatment of rats with Gunmathi Chooranam prevented gastric ulcerogenesis significantly. But it is seemed to be less efficient than standard drug. The result of the present study substantiates the traditional claim that the Gunmathi Chooranam possess antiulcer activity. The results of the present study suggest that the Gunmathi Chooranam may be beneficial in the treatment

of gastric lesions. Further studies to identify the active moieties and elucidation of the mechanism of action are recommended.

Table 1 — Effect of Gunmathi Chooranam on total and free acidity, gastric volume and ulcer index

Groups	Total acidity (mEq/l)	Free aciditly (mEq/l)	Gastric Volume (ml/100g)	Ulcer index (% Ulcer protection)
CMC control	**172±2.28**	**115.11±0.48**	**2.9±0.05**	**1.15±0.02**
Control (Pyloric ligated)	**251±2.84**	**173.23±0.52**	**4.76±0.04**	**5.00±0.05**
Gunmathi Chooranam (125 mg/kg)	**176±2.12****	**120.12±0.74****	**3.00±0.03****	**2.37±0.06** (52.60)**
Gunmathi Chooranam (250mg/kg)	**185±3.07****	**148.16±0.54****	**3.51±0.03****	**1.79±0.05** (64.20)**
Gunmathi Chooranam (500mg/kg)	**197±2.88****	**132.10±0.77****	**3.33±0.03****	**1.68±0.05**(66.40)**
Ranitidine (100mg/kg)	**162±2.19****	**123.08±0.53****	**2.68±0.07****	**1.25±0.05** (75.00)**

***P values <0.05 as compared to ligation control; Values are the mean ± S.E.M. of six rats /treatment. Significance *p <0.05, **p<0.01 Vs Control**

ACUTE AND SUB ACUTE TOXICITY STUDY OF GUNMATHI CHURNAM

PRINCIPLE OF THE TEST

It is the principle of the test that based on a stepwise procedure with the use of a minimum number of animals per step, sufficient information is obtained on the acute toxicity of the test substance to enable its classification. The substance is administered orally to a group of experimental animals at one of the defined doses. The substance is tested using a stepwise procedure, each step using three animals of a single sex. Absence or presence of compound-related mortality of the animals dosed at one step will determine the next step, i.e.; − no further testing is needed, − dosing of three additional animals, with the same dose − dosing of three additional animals at the next higher or the next lower dose level. The method will enable a judgement with respect to classifying the test substance to one of a series of toxicity classes.

Preparation of animals:

The animals are randomly selected, marked to permit individual identification, and kept in their cages for at least 5 days prior to dosing to allow for acclimatization to the laboratory conditions.

Preparation of doses:

In general test substances should be administered in a constant volume over the range of doses to be tested by varying the concentration of the dosing preparation. Where a liquid end product or mixture is to be tested however, the use of the undiluted test substance, i.e. at a constant concentration, may be more relevant to the subsequent risk assessment of that substance, and is a requirement of some regulatory authorities. In either case, the maximum dose volume for administration must not be exceeded. The maximum volume of liquid that can be administered at one time depends on the size of the test animal.

In rodents, the volume should not normally exceed 1mL/100g of body weight: however in the case of aqueous solutions 2 mL/100g body weight can be considered. With respect to the formulation of the dosing preparation, the use of an aqueous solution/suspension/emulsion is recommended

wherever possible, followed in order of preference by a solution/suspension/emulsion in oil (e.g. corn oil) and then possibly solution in other vehicles. For vehicles other than water the toxicological characteristics of the vehicle should be known. Doses must be prepared shortly prior to administration unless the stability of the preparation over the period during which it will be used is known and shown to be acceptable.

Number of animals and dose levels:

Three animals are used for each step. The dose level used as the starting dose was selected from one of four fixed levels, 5, 50, 300 and 2000 mg/kg body weight. The available information suggests that mortality is likely at the highest starting dose level 2000 mg/kg body weight, so the trial or limit test was conducted. Even though there is inadequate information on the test substance, hence for animal welfare reasons the starting dose of 300mg/kg body weight was selected. The time interval between treatment groups is determined by the onset, duration, and severity of toxic signs.

The test substance-related mortality was not produced in animals, so further testing at the next lower level need not be carried out.

OBSERVATIONS:

Animals are observed individually after dosing at least once during the first 30 minutes, periodically during the first 24 hours, with special attention given during the first 4 hours, and daily thereafter, for a total of 14 days, except where they need to be removed from the study and humanely killed for animal welfare reasons or are found dead. All observations are systematically recorded with individual records being maintained for each animal. Observations include changes in skin and fur, eyes and mucous membranes, and also respiratory, circulatory, autonomic and central nervous systems, and somatomotor activity and behaviour pattern. Attention was directed to observations of tremors, convulsions, salivation, diarrhoea, lethargy, sleep and coma. The principles and criteria summarised in the Humane Endpoints Guidance Document taken into consideration. Animals found in a moribund condition and animals showing severe pain or enduring signs of severe distress was humanely killed. When animals are killed for humane reasons or found dead, the time of death should was recorded.

Body weight:

Individual weight of animals was determined before the test substance was administered, and at least weekly thereafter. Weight changes was calculated and recorded. At the end of the test surviving animals were weighed and humanely killed.

Pathology:

All test animals were subjected to gross necropsy. All gross pathological changes were recorded for each animal. Microscopic examination of organs showing evidence of gross pathology in animals surviving 24 or more hours also was considered.

Data and reporting:

All data were summarised in tabular form, showing for each test group the number of animals used, the number of animals displaying signs of toxicity, the number of animals found dead during the test or killed for humane reasons, time of death of individual animals, a description and the time course of toxic effects and reversibility, and necropsy findings.

TOXICITY STUDIES OF GUNMATHI CHURNAM

Administration of doses:

Gunmathi Churanam suspended in 0.5% CMC with vigourous mixing and was administered to the groups of mice in a single oral dose by gavage using a feeding needle. The control group received an equal volume of the vehicle. Animals were fasted prior to dosing. Following the period of fasting, the animals were weighed and then the test substance was administered. After the substance has been administered, food was withheld for a further 3-4 hours. The principle of laboratory animal care was followed. Observations were made and recorded systematically and continuously observed as per the guideline after substance administration. The visual observations included skin changes, mobility, aggressively, sensitivity to sound and pain, as well as respiratory movements. They were deprived of food, but not water 16–18 h prior to the administration of the test suspension. Finally, the number of survivors was noted after 24 h and these animals were then maintained for a further 14 days and observations made daily. The toxicological effect was assessed on the basis of mortality.

Test substance and Vehicle:

The test substance is partially soluble in water but the particle size is large and so the rapid settling was observed. Hence in order to ensure the uniformity in drug distribution in the medium the suspension was made with 0.5% CMC solution and it was found suitable for dose accuracy.

Test animals and Test conditions:

Sexually mature either sex albino mice (28-35g) were obtained from the animal laboratory of the School of Pharmaceutical sciences, Vels University. All the animals were kept under standard environmental condition (27±2°C). The animals had free access to water and standard pellet diet (Sai meera foods, Bangalore). Mice were deprived of food but not water (16-18 h) prior to administration of the Gunmathi Churanam. The principles of laboratory animal care were followed and the Department's ethical committee approved the use of the animals and the study design.

REPEATED DOSE 28-DAY SUBACUTE ORAL TOXICITY STUDY OF GUNMATHI CHURANAM IN RATS

Test Substance: *Gunmathi Churanam*

Animal Source: Vels Animal house

Animals: Male and Female wistar Rats

Age: 6-8 weeks

Body Weight on Day 0: Males: Mean 110.25g Females: Mean 108.42g

Acclimatization: Seven days prior to dosing.

Veterinary examination: Prior to and at the end of the acclimatization period.

Identification of animals: By cage number, animal number and individual marking on fur.

Diet: Pelleted feed supplied by Sai meera foods Pvt Ltd, Bangalore

Water: Aqua guard portable water in polypropylene bottles *ad libitum.*

Housing & Environment: The animals were housed in Polypropylene cages provided with bedding of husk.

Housing temperature between 20^{o} & 24°C,

Relative humidity between 30% and 70%,

Air changes 10 to 15 per hour and

Dark and light cycle each of .12 hours.

Justification for Dose Selection:

As stated in results of acute toxicity studies in wistar rats indicated that *Gunmathi Churanam* was non toxic upto the dose level of 5000 mg/kg body weight observed after 48 hours of oral drug treatment. On the basis of these results, the doses selected for the study was 100mg/kg, 200 mg/kg and 400mg/kg body weight. The oral route was selected for use because oral route is considered to be a proposed therapeutic route.

Preparation and administration of dose:

Gunmathi Churanam was suspended in CMC in distilled water to obtain concentrations of 200mg/ml. It was administered to animals at the dose levels of 100mg/kg, 200mg/kg and 400mg/kg in the dose volume of 10mL/kg. The test substance suspensions were freshly prepared every two

days once for 28 days. The control animals were administered vehicle only. Administration was by oral (gavage), once daily for 28 consecutive days.

Randomization, Numbering and Grouping of Animals:

Six rats were in each group randomly divided into four groups for dosing up to 28 days. Animals were allowed acclimatization period of 7 days to laboratory conditions prior to the initiation of treatment. Each animal was fur marked with picric acid. The females were nulliporous and non-pregnant.

OBSERVATIONS:

Experimental animals were kept under observation throughout the course of study for the following:

(i) ***Body Weight:*** Weight of each rat was recorded on day 0, at weekly intervals throughout the course of study and at termination to calculate relative organ weights. From the data, group mean body weights and percent body weight gain were calculated. (Table-2)

(ii) ***Food and water Consumption:*** The quantity of food consumed by groups consisting of six animals of for different doses was recorded at weekly interval. Food consumed per animal was calculated for control and the treated dose groups. (Table-3&4)

(iii) ***Clinical signs***: All animals were observed daily for clinical signs. The time of onset, intensity and duration of these symptoms, if any, were recorded.

(iv) ***Mortality:*** All animals were observed twice daily for mortality during entire course of study.

(v) ***Ophthalmoscopy***: The eyes of experimental animals in control as well as treated groups given different dose levels were examined prior to the initiation of the dosing and in 4^{th} and the 6^{th} week of the study. Eye examination was carried out using a hand slit lamp after induction of mydriasis with Atropine sulphate solution.

(vi) ***Functional Observations***: At the end of the 4^{th} week exposure, 'sensory reactivity' to graded stimuli of different types (auditory, visual and proprioceptive stimuli), 'motor reactivity' and 'grip strength' were assessed.

***Laboratory Investigations*:** Following laboratory investigations were carried out on day 29 in animals' fasted over-night. Blood samples were collected from orbital sinus using sodium heparin (200IU/ml) for Blood chemistry and potassium EDTA (1.5 mg/ml) for Haematology as anticoagulant. Blood samples were centrifuged at 3000 r.p.m. for 10 minutes.

***Haematological Investigations*:** Haematological parameters were determined using Haematology analyzer. (Table-5)

***Biochemical Investigations*:** Biochemical parameters were determined using auto-analyzer. (Table-6, 7 & 8)

Urine analysis: Urine samples were collected in week 4 and in week 6 and for estimation of normal parameters. The estimations were performed using appropriate methodology. (Table-9)

Necropsy: All the animals were sacrificed on day 29. Necropsy of all animals was carried out and the weights of the organs including liver, kidneys, adrenals, spleen, brain, heart, uterus and testes/ovaries were recorded. The results are shown in Table-10 .

Histopathology: Tissue samples of organs from control and treated animals at the highest dose level were preserved in 10% formalin. The organs included brain, heart, kidneys, liver, lungs, spleen, stomach, and uterus of the animals were preserved they were subjected to histopathological examination.

Statistical analysis: findings such as clinical signs of intoxication, body weight changes, food consumption, haematology and blood chemistry were subjected to One-way Anova followed by dunnet't' test using a computer software programme -INSTAT-V3 version.

RESULTS:

Clinical signs:

Animals were not shown any significant toxic clinical signs during the dosing period of 28 days.

Mortality: All animals from control and all the treated dose groups survived throughout the dosing period of 28 days and it was found no animal dead after 28 days of treatment in high dose.

Body weight: Results of body weight determination of animals of control and different dose groups exhibited comparable body weight gain throughout the dosing period of 28 days.

Food consumption: During dosing period, the quantity of food consumed by animals from different dose groups was found to be comparable and normal with that of control animals.

Ophthalmoscopy: Ophthalmoscopic examination of animals in control and test product– treated groups did not reveal any major and remarkable abnormality.

Functional Observations: These tests conducted on the experimental animals at termination and recorded did not reveal any abnormalities.

Urine analysis: Urine analysis data (Table 9) of control group and treated group of animals determined in week 4 and animals in week 6 did not reveal any abnormalities.

Organ Weight: Group Mean Relative Organ Weights (%of body weight) are recorded in Table-10. Comparison of organ weights of treated animals with respective control animals on day 29 was found to be comparable.

Necropsy: Gross pathological examination of animals in control as well as the treated groups did not reveal any abnormalities.

Haematological investigations: The results of haematological investigations (Table 5) conducted on day 29, revealed following significant changes in the values of different parameters investigated when compared with those of respective controls; However, the increase or decrease in the values obtained was within normal biological and laboratory limits. A slight increase in total RBC count values were obtained for animals in the dose group of 200 and 400 mg/kg ($P<0.05$). Decreased values of platelets ($P<0.05$) were observed for animals in dose groups administered 400mg/ kg body weight of *Gunmathi Churanam* sacrificed on day 29.

Biochemical Investigations: Results of Biochemical investigations conducted on days 29 and recorded in Table 6,7,8 revealed the following significant changes in the values of different parameters studied when compared with those of respective controls; however, the values obtained were within normal biological and laboratory limits. Blood Urea Nitrogen showed reduced levels in animals in 400mg/kg dose group ($P<0.05$), Protein level is elevated in animals of 200 and 400 mg/kg dose group ($P<0.05$). Aspartate Amino transferase levels slightly decreased in animals of 200 and 400mg/kg group ($P<0.01$).

DISCUSSION:

1) All the animals from control and all the treated dose groups up to 400 mg/kg survived throughout the dosing period of 28 days.

2) No signs of major or significant intoxication were observed in animals from lower to higher dose groups during the dosing period of 28 days.

3) Animals from all the treated dose groups exhibited comparable body weight gain with that of controls throughout the dosing period of 28 days.

4) Food consumption of control and treated animals was found to be comparable throughout the dosing period of 28days.

5) Ophthalmoscopic examination, conducted prior to and at the end of dosing period on animals from control and all the treated dose groups did not reveal any abnormality.

6) Haematological analysis conducted at the end of the dosing period on day 29, revealed no significant abnormalities attributable to the treatment.

7) Biochemical analysis conducted at the end of the dosing period on day 29, revealed no remarkable abnormalities attributable to the treatment.

8) Functional observation tests conducted at termination revealed no abnormalities.

9) Urine analysis, conducted at the end of the dosing period in week 4 and at the end of recovery period in week 6, revealed no abnormality attributable to the treatment.

10) Organ weight data of animals sacrificed at the end of the dosing period was found to be comparable with that of respective controls.

11) Gross pathological examination did not reveal any abnormality.

12) Histopathological examination did not reveal any abnormality.

CONCLUSION:

Based on these findings, no toxic effect was observed upto 400mg/kg of *Gunmathi Churanam treated* via oral route over a period of 28 days. So, it can be concluded that the *Gunmathi Churanam* can be prescribed for therapeutic use in human with the dosage recommendations of upto 400mg/kg. body weight p.o.

Table 1: Dose finding experiment and its behavioral Signs of Toxicity

No	Dose mg/kg	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1.	**5**	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2	**50**	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3	**300**	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
4	**2000**	+	-	-	+	-	+	+	-	-	-	-	-	+	-	-	-	-	-	-	-

1. Alertness 2. Aggressiveness 3. Pile erection 4. Grooming 5. Gripping 6. Touch Response 7. Decreased Motor Activity 8. Tremors 9. Convulsions 10. Muscle Spasm 11. Catatonia 12. Muscle relaxant 13. Hypnosis 14. Analgesia 15.Lacrimation 16. Exophthalmos 17. Diarrhoea 18. Writhing 19. Respiration 20. Mortality

Table 2. Body wt (g) of albino rats exposed to *Gunmathi Churnam* for 28days.

Dose (mg/kg/day)	Days				
	1	**7**	**14**	**21**	**28**
Control	112.54±5.05	110.56±6.00	114.82±5.25	118.05±8.11	123.00±5.69
100	117.07±6.31	119.21±5.15	122.19±5.76	125.64±10.00	126.56±6.08
200	114.26±5.44	118.13±6.88	121.08±6.15	124.18±7.14	127.37±8.14
400	112.13±7.27	115.62±5.34	118.12±6.98	121.02±6.33	124.51±7.32

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. N=6.

Table 3. Food (g/day) intake of albino rats exposed to *Gunmathi Churnam* for 28days.

Dose (mg/kg/day)	Days (gms/rats)				
	1	**7**	**14**	**21**	**28**
Control	44.05±2.99	44.88±2.15	46.14±2.12	45.08±2.55	47.52±3.21
100	42.27±2.78	45.33±2.46	46.46±2.66	49.10±2.96	48.15±3.00
200	40.38±2.19	42.04±2.59	44.62±2.47	45.86±3.16	46.00±3.08
400	43.61±2.56	45.28±2.83	46.11±2.85	45.17±2.00	45.00±3.13

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. N=6.

Table 4. Water (ml/day) intake of male and female albino rats exposed to *Gunmathi Churnam* for 28days.

Dose (mg/kg/day)	Days(ml/rat)				
	1	**7**	**14**	**21**	**28**
Control	55.00±2.89	52.07±3.38	55.28±3.18	52.37±3.10	51.20±3.29
100	52.18±2.46	50.22±3.00	45.23±4.00	46.15±3.04	40.55±2.88
200	49.74±2.88	40.16±3.78	40.87±3.39	42.10±2.98	41.10±3.27
400	52.31±3.50	54.67±3.05	51.26±3.88	48.20±3.10	45.23±3.65

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. N=6.

Table 5. Hematological parameters after 28days treatment with *Gunmathi Churnam* in rats.

Parameter	Control	100 mg/kg	200 mg/kg	400 mg/kg
Red blood cell (mm^3)	8.06±0.75	7.98±0.64	8.15±0.59	9.03±0.52
HB (%)	14.12±0.32	15.12±0.39	14.56±0.42	15.00±0.48
Leukocyte ($x10^6$/mL)	10228±115.96	10385±207.24	10305±234.15	10366±219.34
Platelets/ul	1442±43.19	1396±38.24	1137±32.22	995±32.46
MCV (gl)	55.46±5.28	53.15±4.27	55.04±5.27	56.14±4.86
DLC (%)				
N	5.66±1.31	5.23±1.27	4.88±0.96	5.15±3.79
L	95.30±2.98	90.80±3.51	93.28±3.62	96.32±3.88
M	2.0±0.33	2.0±0.38	2.24±0.28	2.32±0.26
E	1.00±0.00	1.0±0.22	1.0±0.11	1.00±0.11
B	0	0	0	0
ESR(mm)	1±00	1±00	1±00	1±00
PCV	46.32±2.56	45.24±2.14	45.00±3.00	45.64±3.22

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. N=6.

Table 6. Effect of treatment with *Gunmathi Churnam* biochemical parameters.

LFT

Dose (mg/kg)	Control	100 mg/kg	200 mg/kg	400 mg/kg
Total Bilirubin (mg/dL)	0.209±0.05	0.212±0.06	0.218±0.05	0.215±0.04
Bilirubin direct (mg/dL)	0.1±0.04	0.1±0.05	0.1±0.04	0.1±0.05
Bilirubin indirect(mg/dL)	0.1±00	0.1±00	0.1±00	0.1±00
ALP (U/L)	382.34±10.16	374.21±12.32	286.38±10.30	294.1±12.32
SGOT (U/L)	168.24±6.21	160.67±6.58	156.38±5.80	154.01±6.57
SGPT(U/L)	46.4±2.34	44.8±3.20	45.08±2.58	46.62±4.19
Total Protein(g/dl)	10.02±1.30	9.17±0.30	8.52±0.27	9.12±0.46
Albumin(g/dl)	3.21±0.25	3.19±0.24	3.46±0.33	3.22±0.12
Globulin(g/dl)	6.02±0.18	5.18±0.26	4.98±0.21	4.86±0.30

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. *vs. control group* N=6.

Table-7 RFT

Dose (mg/kg)	Control	100 mg/kg	200 mg/kg	400 mg/kg
Urea(mg/dL)	55.42±1.63	54.34±3.56	55.2±2.14	53.88±1.36
Creatinine (mg/dL)	0.78±0.05	0.76±0.05	0.78±0.06	0.76±0.05
Uric acid (mg/dL)	1.6±0.12	1.6±0.18	1.6±0.16	1.6±0.14
Na m.mol	140.78±5.26	141.2±5.00	141.12±5.22	141.10±5.00
K m.mol	20.20±2.88	19.45±1.68	20.0±1.46	20.15±2.02
Cl m.mol	100.05±4.24	101.20±5.21	99.88±4.82	101.04±5.16

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. *vs. control group* N=6.

Table-8. Lipid Profile

Dose (mg/kg)	Control	100 mg/kg	200 mg/kg	400 mg/kg
Total cholestrol(mg/dL)	40.68±2.57	41.12±2.46	40.12±3.20	42.05±3.00
HDL(mg/dL)	13.02±1.44	13.25±1.77	13.00±1.45	13.20±2.31
LDL(mg/dL)	42.00±2.83	44.05±3.61	42.31±3.90	43.28±3.32
VLDL(mg/dl)	16.38±2.62	15.82±2.34	16.00±1.64	15.06±1.24
Triglycerides (mg/dl)	88.24±3.00	85.16±2.22	86.23±3.24	88.44±2.78
TC/HDL ratio (g/dl)	3.56±0.24	3.80±0.28	3.75±0.30	3.53±0.25
Blood glucose(mg/dl)	125.35±6.78	126.90±4.25	126.13±5.66	124.11±2.45

Table-9 Urine Analysis

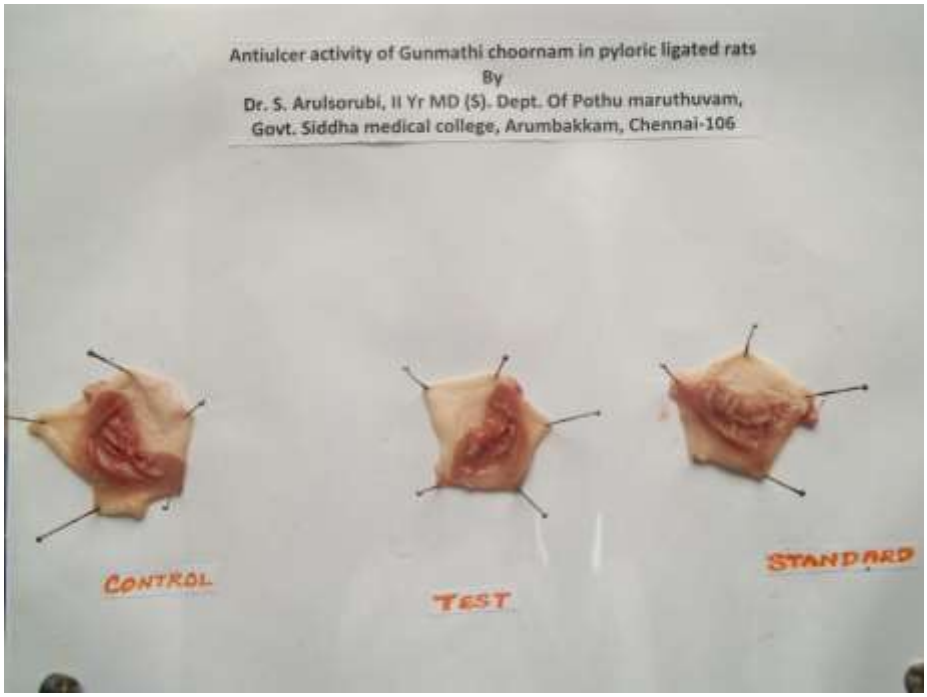
Parameters	Control	100 mg/kg	200 mg/kg	400 mg/kg
Colour	Yellow	Yellow	Yellow	Yellow
Transparency	Clear	Slightly turbid	Slightly cloudy	Slightly turbid
Specific gravity	1.010	1.010	1.010	1.010
PH	>7.2	>8.0	>8.0	>9.0
Protein	Nil	3+	3+	3+
Glucose	Nil	Nil	Nil	Nil
Bilirubin	-ve	-ve	-ve	-ve
Ketones	-ve	+ve	+ve	+ve
Blood	Absent	Absent	Absent	Absent
Urobilinogen	Normal	Abnormal	Abnormal	Abnormal
Pus cells	0-cells/HPF	1-cell/HPF	2-cells/HPF	1-cell/HPF
RBCs	Nil	Nil	0-1cells/HPF	Nil
Epithelial cells	Nil	1-cell/HPF	Nil	1-cell/HPF
Crystals	Nil	Nil	Nil	Nil
Casts	Nil	Nil	Nil	Nil
Others	Bacteria seen	Bacteria seen	Bacteria seen	Bacteria seen

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. *vs. control group* N=6.

Table 10. Effect of oral administration of a *Gunmathi Churnam* on organ weight

Dose (mg/kg)	Control	100 mg/kg	200 mg/kg	400 mg/kg
Liver (g)	5.20±0.17	5.32±0.15	4.92±0.12	5.15±0.18
Heart (g)	0.62±0.04	0.62±0.05	0.59±0.04	0.58±0.04
Lung (g)	1.49±0.06	1.44±0.14	1.36±0.24	1.52±0.15
Spleen (g)	0.65±0.05	0.68±0.04	0.66±0.04	0.65±0.05
Ovary (g)	1.69±0.14	1.78±0.15	1.68±0.18	1.76±0.15
Testes (g)	1.48±0.10	1.45±0.12	1.46±0.15	1.49±0.15
Brain (g)	1.56±0.15	1.58±0.13	1.56±0.14	1.53±0.14
Kidney (g)	0.73±0.04	0.71±0.04	0.70±0.04	0.72±0.05
Stomach (g)	1.36±0.12	1.34±0.10	1.38±0.11	1.35±0.12

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01 *vs control* N=6.


Antiulcer activity of Gunmathi choornam in pyloric ligated rats
By
Dr. S. Arulsorubi, II Yr MD (S). Dept. Of Pothu maruthuvam,
Govt. Siddha medical college, Arumbakkam, Chennai-106
CONTROL
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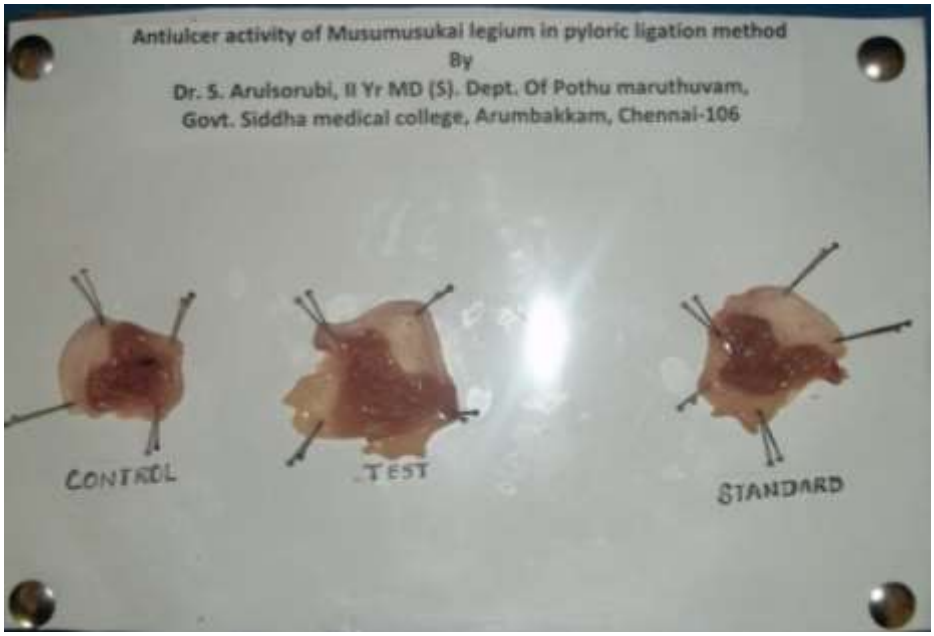
STANDARD


Antiulcer activity of Musumusukai legium in pyloric ligation method
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CONTROL
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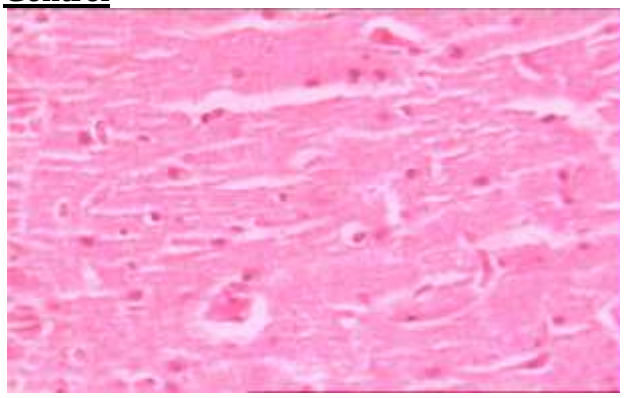
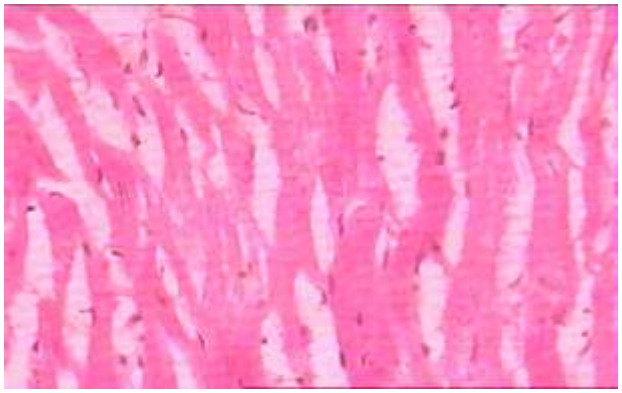
Histopathology

Antiulcer Activity of Gunmathi choornam & Musumusukai Laheam

BRAIN	HEART
Control	
	
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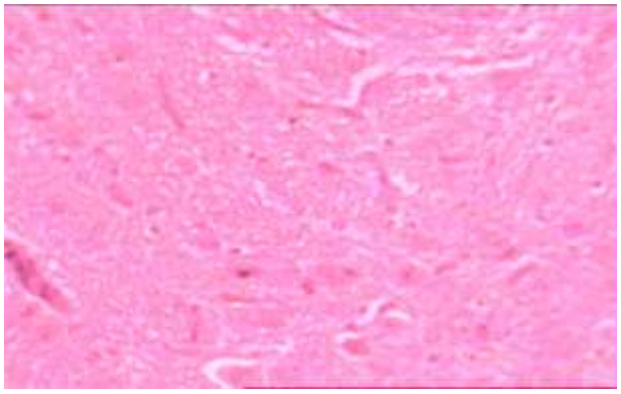	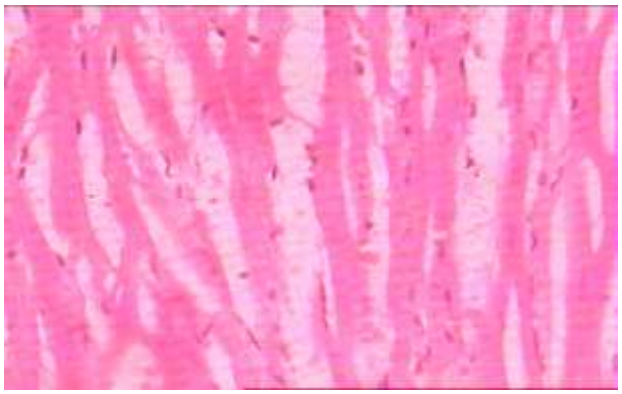
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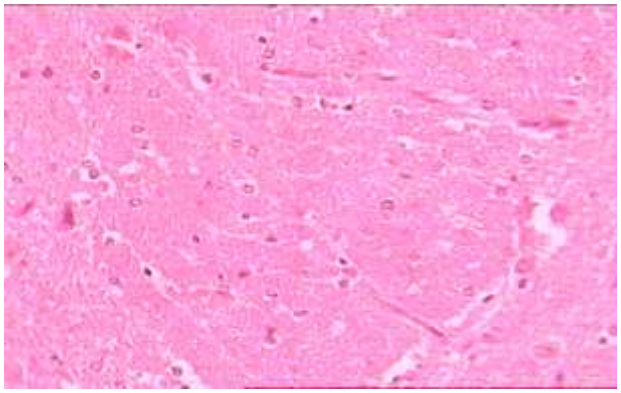	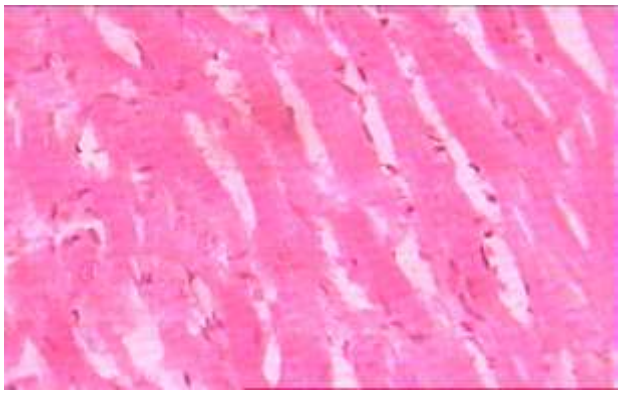

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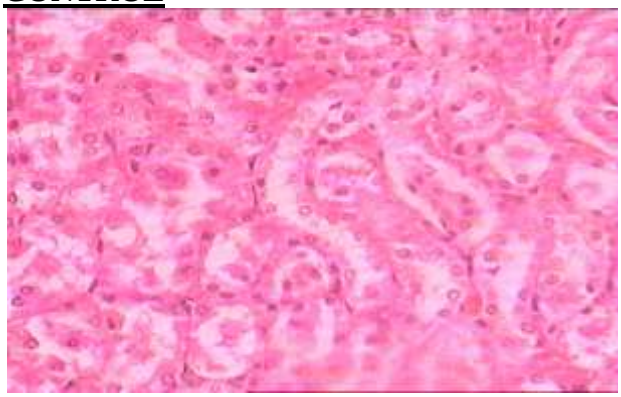	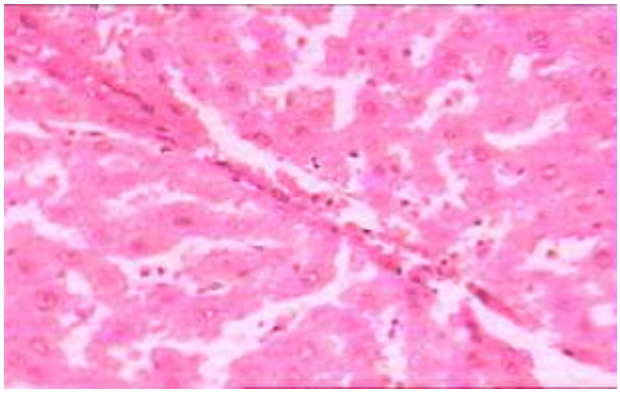
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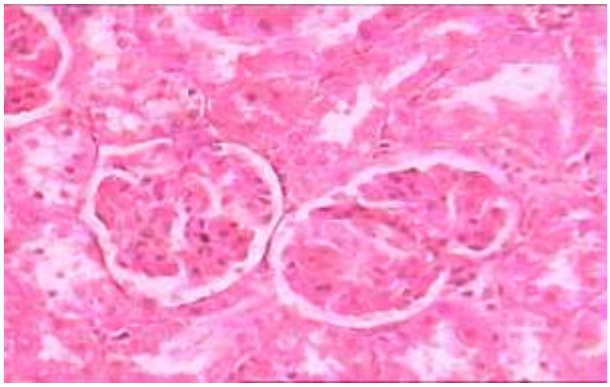	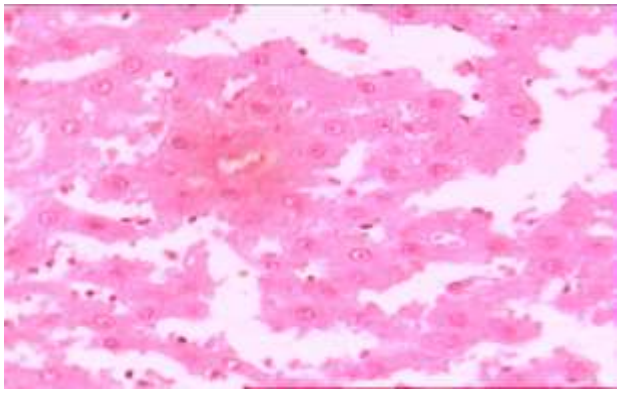
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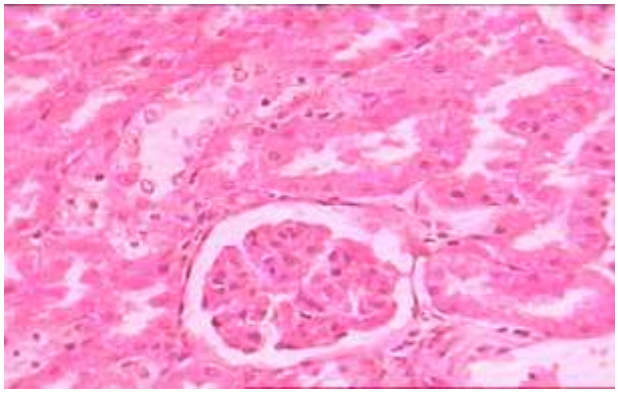	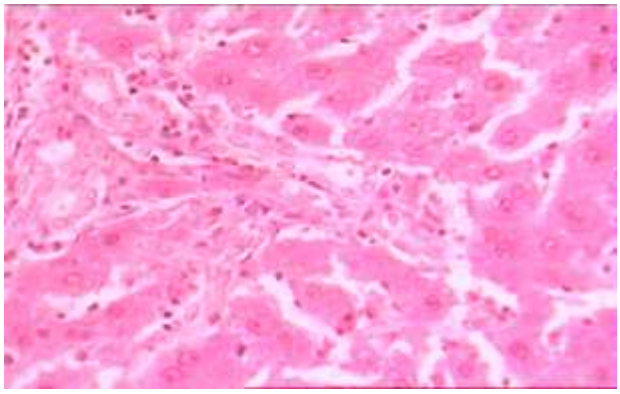

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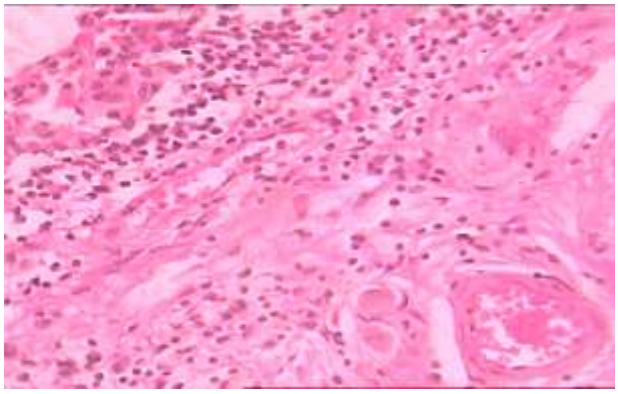	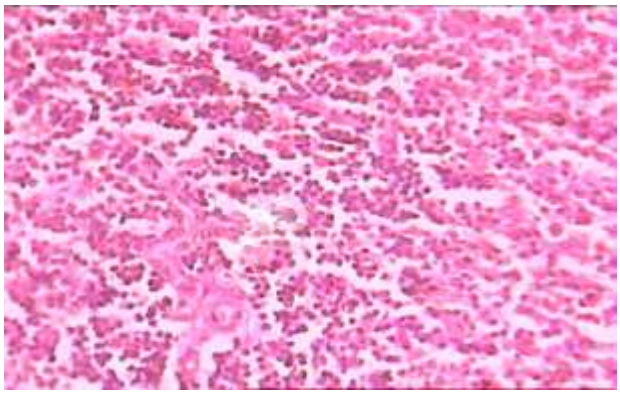
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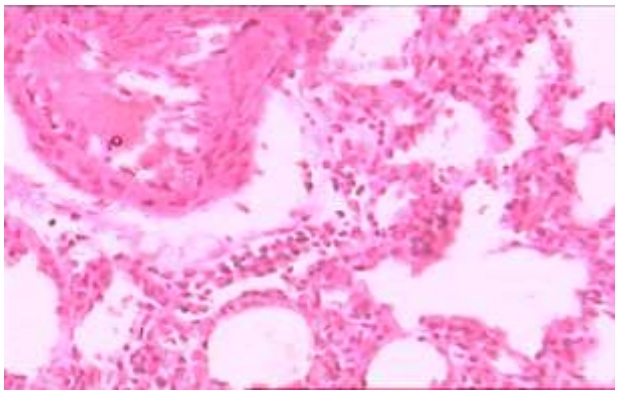	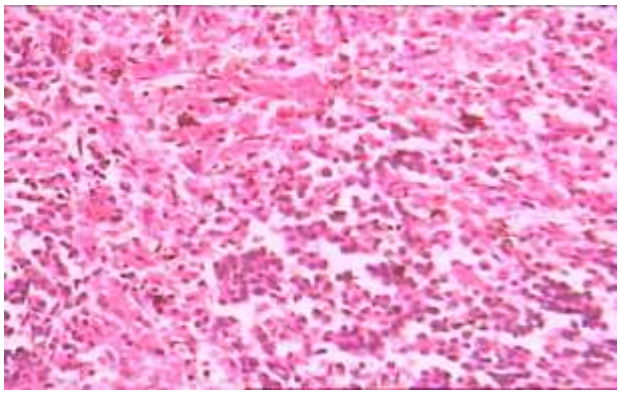
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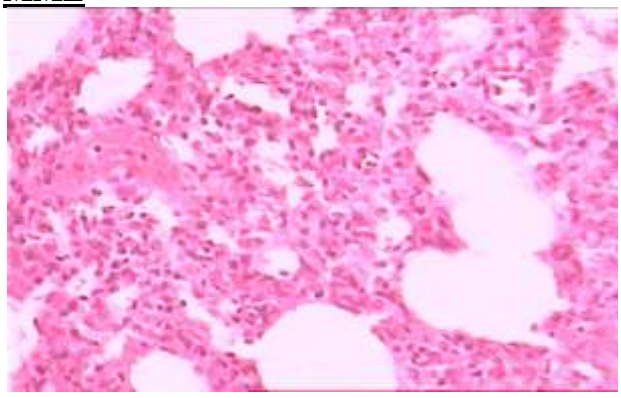
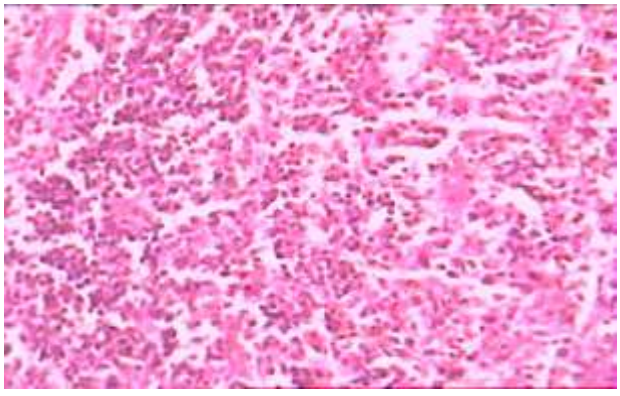
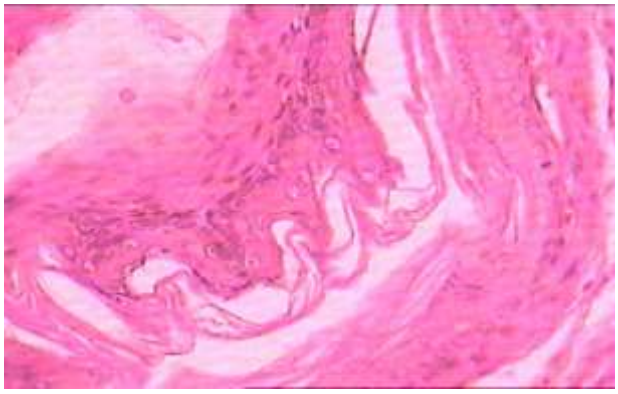
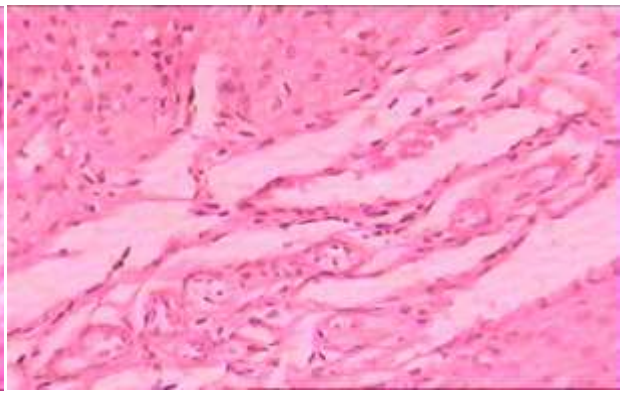
	

STOMACH	UTERUS
CONTROL	
	
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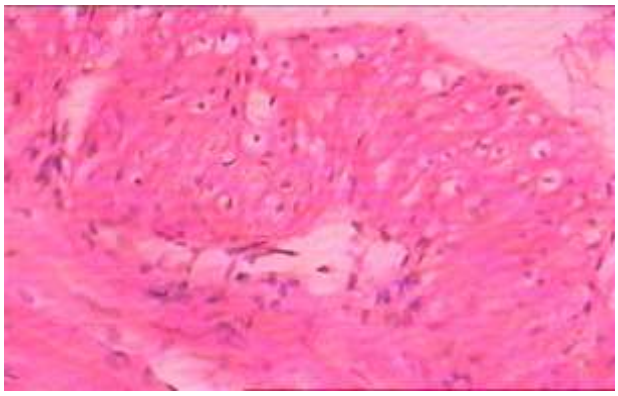	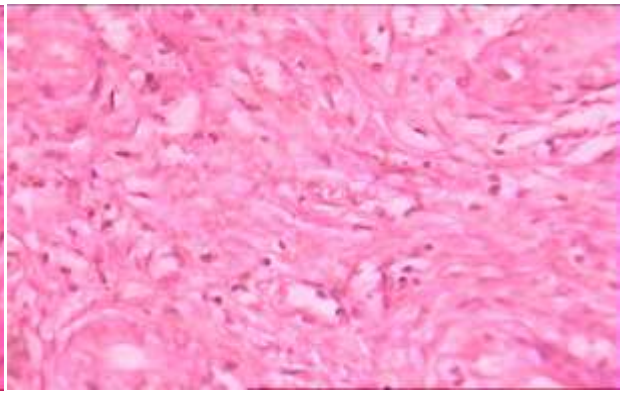
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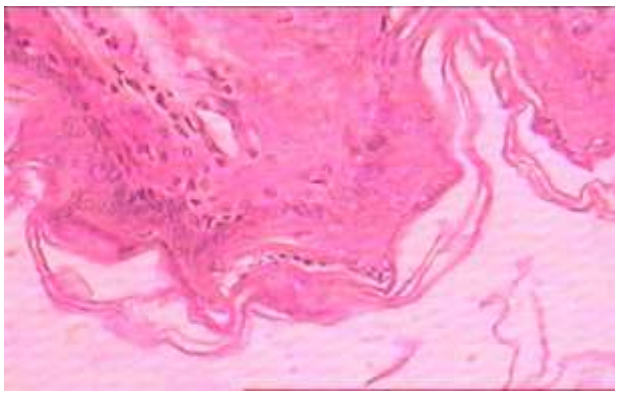	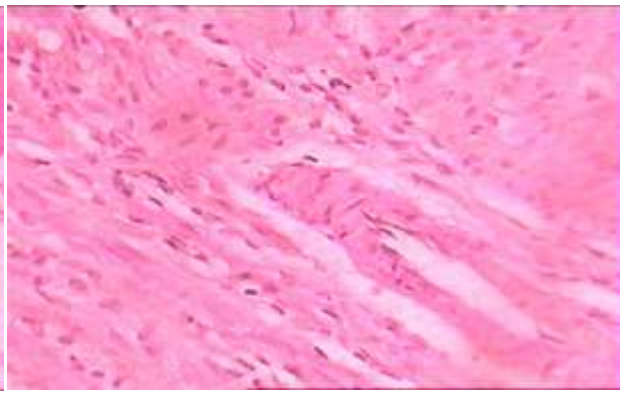

ANTIULCER ACTIVITY OF MUSUMUSUKAI LEGIUM BY PYLORIC LIGATION METHOD IN RATS

Drug and Preparation of Stock solution

The Musumusukai legium was soluble but solid mass settling was observed so as to get uniformity in concentration the solution was converted into suspension form using 2% gum acacia. The final stock solution for this study was 200mg/ml.

Acute oral toxicity study

Acute oral toxicity study of Musumusukai legium was carried out in Swiss albino mice of both sexes (28-32g) according to OECD guidelines no 423.

Antiulcer activity-Grouping and treatment

Pyloric ligation induced gastric ulceration.

Albino rats of either sex were divided into five groups of six animals each. Animals were fasted for 24 h before the study, but had free access to water.

Group I: treated with vehicle (2ml/kg, p.o.) and was kept as control.

Group II: treated with Ranitidine (100mg/kg, p.o.) and was kept as standard.

Group III: treated with the Musumusukai legium (100mg/kg, p.o.).

Group IV: treated with the Musumusukai legium (200mg/kg, p.o.).

Group V: treated with the Musumusukai legium (400mg/kg, p.o.).

RESULTS

Acute oral toxicity study

Swiss albino mice of both the sexes treated with Musumusukai legium did not show any behavioral changes, toxic reaction or mortality. It was found to be safe at the dose of 2000mg/kg. LD_{50} of the Musumusukai legium was found to be >2000mg/kg.

Pyloric ligation induced gastric ulceration

In pylorus ligation induced ulcer model a significant increase in gastric volume (4.76±0.04), free acid (173.23±0.52mEq/L) and total acid (251±2.84mEq/L) were noted. Standard drug ranitidine 100mg/kg treatment has significantly reduced ulcer (0.16±0.16) (UI-1.25), gastric volume (2.68±0.07ml), free acid (123.08±0.53mEq/L) and total acid (162±2.19mEq/L). In pylorus ligation induced ulcer model both 200 and 400 except with low dose 100 mg/kg other two doses i.e, medium and high have significantly reduced the ulcer number, ulcer index and the ulcer formation (30% and 62%) is significantly reduced. Similar to the above a significant reduction in gastric volume, free and total acidity is noted with medium and high doses but not with the low doses of Musumusukai legium.

DISCUSSION

In the present study, we have indicated that the Musumusukai legium has an effective antisecretory and anti-ulcer activity against pyloric ligation-induced gastric ulcers. The examination of acute toxicity (LD50) carried out on mice indicated that Musumusukai legium has no toxicity when administered orally (up to 5g/kg). The gastric protective effect of the Musumusukai legium may be related to an antacid effect or cytoprotective properties. The cytoprotective action showed that the effect of Musumusukai legium is not only a simple acid neutralizing activity but the Musumusukai legium has a cytoprotective effect against the gastric mucosa in the rats.

The etiology of peptic ulcer is unknown in most of the cases, yet it is generally accepted that it results from an imbalance between aggressive factors and the maintenance of mucosal integrity through the endogenous defence mechanisms. Musumusukai legium- a siddha drug used in the present study to evaluate the anti-ulcerogenic effect in pylorus ligation induced ulcers in rats.

The causes of gastric ulcer pyloric ligation are believed to be due to stress induced increase in gastric hydrochloric acid secretion and/or stasis of acid and the volume of secretion is also an important factor in the formation of ulcer due to exposure of the unprotected lumen of the stomach to the accumulating acid. Pylorus ligation induced ulcers are due to auto digestion of the gastric mucosa and breakdown of the gastric mucosal barrier. These factors are associated with the development of upper gastrointestinal damage including lesions, ulcers and life threatening perforation and hemorrhage.

CONCLUSION

The antiulcer property of Musumusukai legium in pylorus ligation model is evident from its significant reduction in free acidity, total acidity, number of ulcers and ulcer index. Musumusukai legium treated animals significantly inhibited the formation of ulcers in the pylorus ligated rats and also decreased both the concentration and increased the pH, it is suggested that Musumusukai legium can suppress gastric damage induced by aggressive factors. The results of the present study suggest that the Musumusukai legium may be beneficial in the treatment of gastric lesions. Further studies to identify the active moieties and elucidation of the mechanism of action are recommended.

Table 1 — Effect of Musumusukai legium on total and free acidity, gastric volume and ulcer index

Groups	Total acidity (mEq/l)	Free aciditly (mEq/l)	Gastric Volume (ml/100g)	Ulcer index (% Ulcer protection)
Normal control	**172±2.28**	**115.11±0.48**	**2.9±0.05**	**1.15±0.02**
Control (Pyloric ligated)	**251±2.84**	**173.23±0.52**	**4.76±0.04**	**5.00±0.05**
Musumusukai legium (100mg/kg)	**232±4.12***	**175.04±1.99**	**4.15±0.03**	**5.06±0.03**
Musumusukai legium (200mg/kg)	**163.20±4.28****	**112.67±2.19****	**3.22±0.04****	**3.46±0.04* (30.08%)**
Musumusukai legium (400mg/kg)	**150.04±3.24****	**123.35±1.44****	**2.86±0.03****	**1.90±0.03** (62%)**
Ranitidine (100mg/kg)	**162±2.19****	**123.08±0.53****	**2.68±0.07****	**1.25±0.05** (75%)**

***P values <0.05 as compared to ligation control; Values are the mean ± S.E.M. of six rats /treatment. Significance *p <0.05, **p<0.01 Vs Control**

TOXICITY STUDIES OF MUSUMUSUKAI LEGIUM

Administration of doses:

Musumusukai legium suspended in 0.5% CMC with vigourous mixing and was administered to the groups of mice in a single oral dose by gavage using a feeding needle. The control group received an equal volume of the vehicle. Animals, test conditions, Randomization, Numbering and Grouping and observations are similar as explained in the toxicological study of Gunmathi Chooranam.

Test substance and Vehicle:

The test substance is freely soluble in water but the cake formation or settling was observed. The suspension was made with 0.5% CMC solution for dose accuracy.

SUBACUTE ORAL TOXICITY STUDY OF MUSUMUSUKAI LEGIUM IN RATS

Justification for Dose Selection:

The results of acute toxicity studies revealed that Musumusukai legium was non toxic upto the dose level of 5000 mg/kg body weight on oral drug treatment. Based on these results, the doses were selected for the sub acute toxicity study as 100mg/kg, 200 mg/kg and 400mg/kg body weight.

Preparation and administration of dose:

Musumusukai legium was suspended in CMC in distilled water to obtain concentrations of 200mg/ml. It was administered to animals at the dose levels of 100mg/kg, 200mg/kg and 400mg/kg in the dose volume of 10mL/kg. The test drug suspensions were freshly prepared for entire duration of study. The control animals were administered vehicle only.

RESULTS:

Clinical signs:

Animals were healthy and active in the total duration of the study and none of the animal showed any significant toxic signs during the dosing period of 28 days.

Mortality: The animals of control group and treated groups survived throughout the study.

Body weight: The body weight of the different groups of test drug treated animals and control were gained gradual increase in body weight during the dosing period.

Food consumption: The food consumption by the animals from different treated groups was found to be comparable and satisfactory.

Ophthalmoscopy: Animals of treated groups did not reveal any significant abnormality.

Functional Observations: The experimental animals did not reveal any abnormalities.

Urine analysis: Urine parameters of drug treated group and control found to be normal.

Organ Weight: Organ weight of test drug treated animals was compared with control group of animals on day 29 was found to be normal.

Necropsy: Pathological examination in control as well as the treated groups did not reveal any abnormalities.

Haematological investigations: The results of haematological parameters conducted on day 29, indicated the slight decrease in the total RBC count in the dose group of 400 mg/kg ($P<0.05$). There was no other major alteration in heamatological parameters in drug treated group except Hb level is slightly increased in 400mg/kg group.

Biochemical Investigations: The results of Biochemical investigations conducted on days 29 revealed that the bilurubin range is modified significantly by the high dose of Musumusukkai legium. Blood Urea Nitrogen is slightly minimized in 400mg/kg dose group but it is not statistically significant. Aspartate Amino transferase levels slightly increased in animals of 400mg/kg group ($P<0.05$). No remarkable changes were identified in the other biochemical parameters in all drug treated group of animals. Rest of the values obtained was within normal biological and laboratory limits.

Histopathology: Animals after 28 days exposure to Musumusukkai legium revealed no significant changes in the vital organs. Microscopic observation of kidney & liver found to be normal, Stomach section indicates no abnormal architecture,. Ovary of rats fed with different concentration of Musumusukkai legium for a period of 28 days suggest no significant tissue damage and were comparable with those of normal control animals.

DISCUSSION:

1) All the animals were survived throughout the dosing period of 28 days.

2) No signs of intoxication were observed from lower to higher dose treated groups in throughout the study.

3) Animals from all the treated dose groups exhibited comparable and appreciable weight gain during the dosing period.

4) Food consumption of control and treated animals was found to be satisfactory.

5) Ophthalmoscopic examination, conducted prior to and at the end of dosing period on animals from control and all the treated dose groups did not reveal any abnormality.

6) Haematological analysis conducted at the end of the dosing period on day 29, revealed no significant abnormalities attributable to the treatment.

7) Biochemical analysis conducted at the end of the dosing period on day 29, revealed no remarkable abnormalities attributable to the treatment.

8) Functional observation tests conducted at termination revealed no abnormalities.

9) In the gross examination of urine of test animals, the volume and intensity of colour is increased in dose dependent manner and epithelial, pus cells and oxalate crystals were present in the urine deposit of the drug treated animals.

10) Histopathological and pathological examination did not reveal any major or harmful abnormality.

CONCLUSION:

During the experimental period, there were no treatment-related effects on the hematological parameters evaluated. As the macroscopic appearance and weight of the kidney was not altered, hence, the possibility of renal injuries could not be confirmed. Administration of Musumusukkai legium did not induced any changes in the biochemical parameters. This observation could indicate that liver function is preserved by oral administration of Musumusukkai legium. In conclusion, this study demonstrated that Musumusukkai legium seems to be destitute of toxic effects, which could be compromise the medicinal use of this medicine.

Table 1: Dose finding experiment and its behavioral Signs of Toxicity

No	Dose mg/kg	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1.	**5**	+	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2	**50**	+	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3	**300**	+	-	-	-	+	+	-	-	-	-	-	-	-	-	+	+	+	-	+	-
4	**2000**	+	-	-	+	+	+	-	-	-	-	-	-	-	-	+	+	+	-	+	-

1. Alertness 2. Aggressiveness 3. Pile erection 4. Grooming 5. Gripping 6. Touch Response 7. Decreased Motor Activity 8. Tremors 9. Convulsions 10. Muscle Spasm 11. Catatonia 12. Muscle relaxant 13. Hypnosis 14. Analgesia 15.Lacrimation 16. Exophthalmos 17. Diarrhoea 18. Writhing 19. Respiration 20. Mortality

Table 2. Body wt (g) of albino rats exposed to *Musumusukai Legium* for 28days.

Dose (mg/kg/day)	Days				
	1	**7**	**14**	**21**	**28**
Control	141.0 ± 9.0	141.0 ± 9.0	143.0 ± 7.5	149.0 ± 7.0	170.0 ± 10.0
100	130.0 ± 10.0	130.0 ± 10.0	127.5 ± 8.5	108.0 ± 2.0	105.0 ± 1.0
200	133.0 ± 13.0	133.0 ± 23.0	135.0 ± 20.5	135.0 ± 20.0	133.0 ± 1.0
400	127.5 ± 7.5	127.5 ± 7.5	132.0 ± 7.0	132.0 ± 3.0	136.5 ± 0.5

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. N=6.

Table 3. Food (g/day) intake of albino rats exposed to *Musumusukai Legium* for 28days.

Dose (mg/kg/day)	Days(gms/rats)				
	1	**7**	**14**	**21**	**28**
Control	39.98±3.18	34.51±2.87	39.98±3.18	38.12±2.80	38.56±3.10
100	36.64±3.92	34.63±2.00	33.28±2.00	30.80±2.51	32.49±2.20
200	49.37±3.00	46.18±2.12	44.19±2.863	44.33±2.17	42.14±3.18
400	40.12±2.48	42.50±2.28	40.12±2.19	38.08±2.00	40.00±2.64

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. N=6.

Table 4. Water (ml/day) intake of male and female albino rats exposed to *Musumusukai Legium* for 28days.

Dose (mg/kg/day)	Days(ml/rat)				
	1	**7**	**14**	**21**	**28**
Control	46.68±2.58	44.15±3.66	45.10±2.15	40.14±2.40	41.0±3.02
100	48.16±2.89	45.19±3.48	42.00±3.76	38.68±1.99	40.30±3.78
200	52.00±3.10	49.11±2.19	44.68±3.72	40.28±2.41	40.00±2.40
400	39.46±2.18	42.12±2.40	40.69±2.35	39.77±2.66	39.31±2.46

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. N=6.

Table 5. Hematological parameters after 28days treatment with *Musumusukai Legium* in rats.

Parameter	Control	100 mg/kg	200 mg/kg	400 mg/kg
Red blood cell (mm^3)	6.71±0.78	6.85±0.44	6.91±9.27	6.88±0.36
HB (%)	15.1±0.66	15.18±0.21	15.06±0.32	15.06±0.23
Leukocyte ($x10^6$/mL)	10816.66±487.51	11383±990.79	11266±504.65	11384±845.22
Platelets/ul	957.66±75.38	935.66±21.83	930.5±53.14	938.5±21.81
MCV (gl)	68.38±4.53	60.48±6.64	60.8±6.14	60.31±5.25
DLC (%)				
N	7±1.67	6.5±1.37	7±1.41	6.66±1.63
L	89.66±1.03	90.33±2.16	90.16±1.47	90.16±3.06
M	1.83±0.40	2±0.89	1.83±0.75	2±0.63
E	1±0.00	1±0.00	1.5±0.54	1±0.00
B	0±0.00	0±0.00	0±0.00	0±0.00
ESR(mm)	1±00	1±0.02	1.5±0.04	1±00
PCV	45.66±2.95	41.33±4.17	42±3.40	42±3.84

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. N=6.

Table 6. Effect of treatment with *Musumusukai Legium* biochemical parameters.

LFT

Dose (mg/kg)	Control	100 mg/kg	200 mg/kg	400 mg/kg
Total Bilirubin (mg/dL)	0.231±0.09	0.233±0.08	0.224±0.02	0.245±0.05
Bilirubin direct (mg/dL)	0.15±0.083	0.15±0.054	0.16±0.08	0.02±0.089
Bilirubin indirect (mg/dL)	0.16±0.081	0.21±0.07	0.23±0.08	0.28±0.07
ALP (U/L)	189±63.56	239±41.35	197.33±56.09	206.33±64.30
SGOT (U/L)	153.33±16.04	156±15.38	159.33±16.23	170.33±11.05
SGPT(U/L)	62.5±15.21	55.5±5.32	57.66±5.39	52.5±5.01
Total Protein(g/dl)	7.98±0.47	8.06±0.39	8.23±0.37	8.08±0.43
Albumin(g/dl)	3.01±0.26	3.16±0.29	3.31±0.27	3±0.35
Globulin(g/dl)	4.96±0.61	4.9±0.56	4.93±0.59	4.91±0.46

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. *vs. control group* N=6.

Table-7 RFT

Dose (mg/kg)	Control	100 mg/kg	200 mg/kg	400 mg/kg
Urea(mg/dL)	58.19±1.56	62.10±2.88	64.19±2.10	64.98±2.80
Creatinine (mg/dL)	0.45 ± 0.050	0.48 ± 0.04	0.45 ± 0.04	0.45 ± 0.06
Uric acid (mg/dL)	1.5±0.10	1.51±0.27	1.51±0.29	1.46±0.23
Na m.mol	138.12±7.30	137.10±5.71	134.15±5.30	132.64±5.07
K m.mol	21.60±2.84	27.83±1.16	28.5±1.87	29.16±1.47
Cl m.mol	99.14±3.18	102.83±3.37	102.5±2.42	103.16±2.13

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. *vs. control group* N=6.

Table-8. Lipid Profile

Dose (mg/kg)	Control	100 mg/kg	200 mg/kg	400 mg/kg
Total cholestrol(mg/dL)	41.98±2.78	38.07±3.28	38.30±4.22	40.66±3.50
HDL(mg/dL)	12.19±1.65	12.6±1.12	13.01±0.27	13.00±1.32
LDL(mg/dL)	32.8±2.88	31.99±2.53	40.08±2.90	38.35±2.10
VLDL(mg/dl)	16.19±2.46	16.60±1.00	17.4±2.81	16.87±5.70
Triglycerides (mg/dl)	82.15±3.38	109.66±16.76	102.33±19.81	108.91±20.34
TC/HDL ratio (g/dl)	3.42±0.21	3.46±0.12	3.84±0.26	4.10±0.28
Blood glucose(mg/dl)	112.16±8.62	109.7±6.50	119.08±4.48	114.5±6.22

Table-9 Urine Analysis

Parameters	Control	100 mg/kg	200 mg/kg	400 mg/kg
Colour	Yellow	Straw Yellow	PaleYellow	Yellow
Transparency	Clear	Clear	Dark	High intense
Specific gravity	1.010	1.010	1.010	1.010
PH	>7.2	7.2	>7.0	>7.2
Protein	Nil	Nil	Nil	Nil
Glucose	Nil	Nil	Nil	Nil
Bilirubin	-ve	-ve	-ve	-ve
Ketones	-ve	-ve	-ve	-ve
Blood	Absent	Absent	Absent	Absent
Urobilinogen	Normal	Normal	Normal	Normal
Pus cells	0-cells/HPF	Nil	4-5cells/HPF	3-5cells/HPF
RBCs	Nil	Nil	Nil	Nil
Epithelial cells	Nil	1-2cells/HPF	1-2cells/HPF	2-3cells/HPF
Crystals	Nil	Nil	Nil	Nil
Casts	Nil	Nil	Nil	Nil
Others	Bacteria seen	Bacteria seen	Bacteria seen	Bacteria seen

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01. *vs. control group* N=6.

Table 10. Effect of oral administration of a *Musumusukai Legium* on organ weight

Dose (mg/kg)	Control	100 mg/kg	200 mg/kg	400 mg/kg
Liver (g)	4.48±0.8	4.66±0.7	4.96±0.10	4.29±0.11
Heart (g)	0.56±0.04	0.58±0.03	0.53±0.04	0.55±0.04
Lung (g)	1.28±0.7	1.12±0.9	1.30±0.05	1.22±0.04
Spleen (g)	0.58±0.05	0.62±0.05	0.58±0.04	0.65±0.04
Ovary (g)	2.12±0.10	2.0±0.06	2.10±0.06	2.0±0.8
Testes (g)	1.36±0.12	1.33±0.10	1.38±0.8	1.36±0.10
Brain (g)	1.43±0.12	1.46±0.8	1.42±0.05	1.46±0.05
Kidney (g)	0.66±0.07	0.64±0.05	0.67±0.05	0.65±0.07
Stomach (g)	1.33±0.9	1.30±0.10	1.29±0.04	1.30±0.12

Values are mean of 6 animals ± S.E.M. (Dunnett's test). *P<0.05; **P<0.01 *vs control* N=6.

ANNEXURE-VI

Proforma of Case Sheet

OP/IP CASE SHEET PEROFORMA

POST GRADUATE DEPARTMENT, POTHU MARUTHUVAM (BRANCH-I)

GOVT.SIDDHA MEDICAL COLLEGE & HOSPITAL, CHENNAI-106.

PROFORMA FOR 'ERIGUNMAM'

OP/IP No	:	OCCUPATION:
WARD No	:	INCOME:
BED No	:	NATIONALITY:
NAME	:	RELIGION:
AGE	:	D.O.A:
SEX	:	D.O.D:
DIAGNOSIS	:	

PERMANENT ADDRESS:

TEMPERORY ADDRESS:

MEDICAL OFFICER

COMPLIANTS AND DURATION

HISTORY OF PRESENT ILLNESS

HISTORY OF PAST ILLNESS

PERSONAL HISTORY/ HABITS

a. Food	Veg	Non Veg
b. Marital status	Single	Married
c. Smoking	Yes	No
d. Drinking	Yes	No

FAMILY HISTORY:

GENERAL EXAMINATION:

Physical built:	Lean	Normal	Obese

Consciousness:

Body weight:

Temperature:

Pallor:

Cyanosis:

Jaundice:

Clubbing:

Pedal Oedema:

Lymph adenopathy:

Jvp:

Pulse rate:

Heart rate:

Respiratory rate:

Blood pressure:

Examination of vital organs:

Heart

Lungs

Abdominal organs

Palpitation:

Tenderness in epigastric region:	present	absent

SIDDHA ASPECTS

Yaakai (udalnilai)

1. Vatham
2. Pitham
3. Kapham
4. Kalappu

Mukkunam

1. Sathuva gunam
2. Raasatha gunam
3. Thamo gunam

Paruva kaalam (seasons)

1. Karr (Aavani-Puratasi) Aug 16-Oct15
2. Koothri (Aypasi-Karthigai) Oct16-Dec15
3. Munpani (Maargazhi-Thai) Dec16-Feb15
4. Pinpaani (Massi-Panguni) Feb 16-Apr15
5. Elavenil (chithirai-Vaikasi) Apr16- Jun 15
6. Mudhuvenil (Aani-Aadi) Jun16-Aug 15

Nilam (places)

1. Kurinchi (Hill area)
2. Mullai (Forest Area)
3. Marutham (Fertile Area)
4. Neithal (Sea Area)
5. Paalai (Desert Area)

Iyamporigal/Pulangal

1. Mei (Sensation)
2. Vaai (Taste)
3. Kann (vision)
4. Mooku (Smell)
5. Sevi (Hearing)

Kanmenthiriyam/Kanmavidayam

1. Kai (Koduthal)
2. Kaal (Nadathal)
3. Vaai (Pesal)
4. Eruvai (Kazthial)
5. Karuvai (Ananthithal)

Mummalam

Malam
Moothiram
Viyarvai

Kosam

1. Annamaya Kosam (paru udambu)
   (yelu udal Thaathukkal)
2. Pranamaya Kosam (Vali udambu)
   (Pranan + Kanmenthiriyam)
3. Manomaya Kosam (Mana udambu)
   (Manam + Gnanethiriyam)
4. Gnanamaya Kosam (Arivu udambu)
   (Puththi + Gnanenthiriyam)
5. Ananthamaya Kosam (Inba udambu)
   (Pranan + Suzhuthi)

Uyir Thathukkal:

Vatham:

1. Pranan
2. Abanan
3. Viyanan
4. Udhanan
5. Samanan
6. Naagan
7. Koorman
8. Kirukaran
9. Devadathan
10. Dhananjayan

Pitham:

1. Analagam
2. Ranjagam
3. Saadhagam
4. Aalosagam
5. Prasagam

Kapham:

1. Avalambagam
2. Kledagam
3. Podhagam
4. Tharpagam
5. Santhigam

Udal Thathukkal:

1. Saaram
2. Senneer
3. Oon
4. Kozhuppu
5. Enbu
6. Moolai
7. Sukkilam / Suronitham

Envagai Thervu:

1. Naa
2. Niram
3. Mozhi
4. Vizhi
5. Sparisam
6. Malam
    a) Niram
    b) Nurai
    c) Erugal
    d) Elagal

Moothiram

1. Neerkuri
    a) Niram
    b) Edai
    c) Manam
    d) Nurai
    c) Enjal
2. Neikuri

Naadi

SIGNS AND SYMPTOMAS OF ERIGUNMAM

Symptoms	**Before treatment**	**After treatment**			
I.PAIN RELATED TO FOOD		7^{th} day	14^{th} day	21^{st} day	30^{th} day
A.Epigastric discomfort a. After meals b. 1 to 2 hours a. meals c. 2 to 4 hours a. meals					
B.Burning sensation in Epigastrium a.Before meals b. 1 to 2 hours a. meals c. 2 to 4 hours a. meals d. Contant.					
C. Site of pain Pain radation a. No radation b. Left shoulder c. Back d. Sides of chest.					
E. Diarrhoea Present Not present If present 1 to 2 hours a.m. 2 to 4 hours a.m.					
F. pain relived by a . food b. antacids c. bed rest d. sidhha drugs e. vomiting f. not relived by all methods					

	Before treatment	**After treatment**			
I. **Appetite** a. Poor b. Moderate c. Normal d. ferocious		7^{th} day	14^{th} day	21^{st} day	30^{th} day
Signs :- Examination Of Abdomen					
1. Tenderness epigastrium a. Present b. Not present					
2.Headache a. Present b. Not present					
3.Rigidity of rectus Abdominus a. Present b. Not present					
4. Visible Gastric Pesistalsis a. Present b. Not present					
5. Palpable mass a. Present b. Not present					

OTHER SIGNS AND SYMPTOMS

	Before treatment	After treatment
1. Constipation		
2. Bloating		
3. General debility		
4. Anorexia		
5. Giddiness		
6. Heaviness Of Head		
7. Cough		
8. Dyspnoea		
9. Urine Coloured		
10. Sweating		
11. Palpitation		
12. Emaciation		
13. Heamatemisis		

Laboratory investigation

BT **AT** **Blood**

Tc

Dc

ESR

Hb

Bl sugar (R)

Bl sugar (F) (PP)

Bl urea

Sr.cholesterol

Urine

Alb

Sug

Dep

Motion

Ova

Cyst

Occult blood

X ray

Barium meal

Endoscopy

USG

Trial drug

Dose

Anubanum

Duration of treatment

Pathiam (do's &don'ts)

Prognosis at the end of the treatment

Medical officer signature

H O D

CONSENT FORM

I certify that I have disclosed all the details about the study of the terms readily Understood by the patients.

Date: **SIGNATURE**

NAME

CONSENT BY THE PATIENT

I have been informed to my satisfaction by the attending physician the purpose of the clinical trial and nature of the drug treatment and follow up including the lab investigation to be performed to monitor and safeguard my body functions.

I am aware of my right to opt out of the trail at any time during the course of the trail without having to give reasons for doing so.

I exercising my free power of choice, here by give consent to be included as a subject is the clinical trial of **GUNMATHI CHOORNAN** and **MUSUMUSUKAI LEGIYAM** for the treatment of **ERIGUNMAM**

Date: **SIGNATURE**

NAME

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

69, Anna Salai, Guindy, Chennai - 32.

DEPARTMENT OF SIDDHA

CERTIFICATE OF PARTICIPATION

This is to certify that Dr. S. ARULSORUBI

has participated as Resource Person / Delegate in the Workshop on

"Research Methodology & Biostatistics" for AYUSH Post Graduates &

Researchers organized by the Dept. of Siddha from 23-08-10 to 27-08-10

Dr. N. Kabilan
Prof. & Head

Dr. Sudha Seshayyan
Registrar i/c

Dr. Mayil Vahanan Natarajan
Vice-Chancellor

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